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PATHOLOGY AND PRACTICE

GEORGE BLUMER, M.D.
NEW HAVEN, CONN.

Medical and surgical diagnosis rests to a large extent on the recognition of the nature and cause of gross changes in structure and their consequences on function.—LUDVIG HEKTOEN.

Any one who has the hardihood to attempt a discussion of such a time-worn subject as the relation of pathology to practice runs the risk of producing something which, in the words of the melancholy Dane, is "stale, flat and unprofitable." And yet on an occasion like this, when we are marking the end of a period, it seems worth while to chance this risk. It is advantageous, no doubt, to take stock from time to time of the situation that confronts us, particularly during a period when both the science and the art of medicine are in a state of flux, as they have been during the last fifty years. The reasons for the rapidly changing conditions that have characterized this period are not far to seek: New diseases have been discovered, old diseases have been restudied by new methods, diseases that were originally local have become world wide through travel, trade or war, and entirely new disciplines, which necessitate new points of view, have developed. In such circumstances it is well to ask ourselves whether our program of medical training is meeting the altered conditions, whether it is a well balanced one, or whether the itch for novelty, for which even scientific human nature is ever greedy, may not have led us to exalt the new and spectacular at the expense of the old and well tried.

It is my purpose in this paper to discuss briefly the importance to the clinician of a sound training in pathology, a doctrine which, in view of the antiquity of recorded medicine, is not such a very old one. It is true that as early as the seventeenth century William Harvey stated that "from a knowledge of pathology the practice and art of healing, and numberless new methods of treatment, will naturally spring." His postmortem studies, which might well have influenced the profession at that time, were unfortunately destroyed by the parliamentary troopers who sacked his chambers in 1642, and it was not until the publication of Morgagni's observations a century later that the leaders in the profession awoke to the possibilities that lay in the combined clinical and pathologic study of disease. While many of those who succeeded Morgagni, Baillie, Bichat, Laënnec, Rokitansky and Bright, for example, accepted and expanded his point of view, it took a long time for it to make a lasting impression on medical education and a still longer time for it to filter through to the consciousness of the mass of the profession. It was not, of course, until Virchow's time, and Virchow did not die until this century, that the development of pathologic histology broadened the field and it was still later that the development of bacteriology and the application of the newer chemical and physical methods still further extended it.

It is obvious from this brief historical survey that pathology now covers a much wider field than it did in its infancy, when it consisted of gross pathology; in its childhood, when it was a combination of morbid pathology and histology, or even in its adolescence when the germ theory extended its scope. Since then clinical pathology, chemical pathology, pathologic physiology and experimental pathology have all developed and have grown apace. It therefore becomes necessary to define more precisely the particular aspects of pathology that are considered of maximum importance in the training and development of good clinicians, for, needless to say, every student of medicine should have some knowledge of the field as a whole.

It has always seemed to me, and I am doubtless influenced by my own training, that the fields of pathology whose cultivation is most essential to the development of the clinician are morbid anatomy and histology. A study of these subjects makes for precision in diagnosis and, after all, the basis of good clinical work is exact diagnosis, without which the prognosticator and the therapeutist are mere empiricists groping in the dark.

Objections have been raised against the view that morbid anatomy and histology are of abiding importance in the training of the clinician mainly on two grounds:

It has been claimed that their study withdraws attention from morbid physiology or, to put it in another way, that the pursuit of morbid anatomy causes overlooking of the general principles of pathology. One can only say in reply to this objection that it presupposes an attitude of mind on the part of pathologists singularly lacking in imagination and insight. How any thoughtful individual engaged in the study of morbid anatomy and histology could avoid pondering over the effect on function of the structural changes that he observes passes my comprehension. Morbid anatomy and histology passed out of the purely descriptive stage of development generations ago; their method is now that of science in general; namely, observation plus induction.

The second common criticism brought against the study of morbid anatomy and histology is that they reveal only end results. This is a half-truth and, as Professor Leacock has epigrammatically remarked, "a half-truth, like half a brick, is always more forcible as an argument than a whole one—it carries further." It needs very little pathologic experience to enable one to realize that in many chronic diseases all stages of a pathologic process are present in the body at the same

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time. Furthermore, the early stages of chronic disease may often be detected and studied in patients dead of accident or acute disease. In acute disease the duration of illness is often so short and the lesions are so clear cut that the "end results" argument carries no weight.

It is difficult for a clinician who feels his indebtedness to his pathologic training to formulate precisely the ways in which it has influenced his clinical career. Pathology, like diagnosis, is based on observation and induction, and it is clear that for this reason it is a preliminary training in method. But, further than this, it gives the student a much wider point of view of disease than is usually obtained in practice. Many diseases which in the living patient appear to have attacked a single organ or tissue are found on the postmortem table to be much more widespread in their effects than the clinician had suspected. Thus pathology gives an insight into disease as a whole and a clinician with a good pathologic training tends, as a result, to visualize what is going on in the entire body. As one writer puts it, "the greater our power of visualizing the underlying pathological process the closer we come to a proper concept of disease." Furthermore, such visualization, based as it is on accurately observed facts, protects the clinician from the very human tendency to theorize and speculate on insufficient data, which has been such a prominent feature of medicine at certain periods, notably the eighteenth century. The value of morbid anatomy and histology as methods of checking the accuracy of diagnosis and showing the clinician his mistakes is so obvious that it need be mentioned only in order to point out the necessity of encouraging their use to the fullest extent. We all learn more from our mistakes than from our successes, but we cannot learn from our mistakes unless we have them demonstrated to us.

If I have succeeded in making it clear that morbid anatomy and histology are of paramount importance in the training of clinicians, certain practical considerations naturally arise. It is pertinent to inquire whether, under existing conditions, sufficient opportunity is afforded to the medical student and the practitioner for the study of pathology. There is no doubt that improvements in the teaching of pathology have been developed in recent years, notably the well conducted clinical-pathologic conferences that exist in some teaching centers and the seminars on tumor pathology that have been developed in many places as the result of the crusade against cancer. In the training of medical students there is still too great a tendency in some places to treat pathology as a preclinical subject rather than as the most important liaison subject between the laboratory and the clinic. During his clinical years the student should be required much more frequently than he is at present to follow his patients to the autopsy room; indeed, a revival of the method long used at Edinburgh and Manchester of requiring students during their clinical training to pursue such a course and to write case reports that coordinate the whole disease picture from both clinical and pathologic points of view is highly desirable. But the main problem does not lie in the teaching hospitals but in the nonteaching ones in which the majority of graduates spend their intern years. It is essential that all such hospitals should have adequate pathologic departments under well trained pathologists and that the members of the house staff should be required to follow their cases to the autopsy room and study the pathologic observations. In these hospitals the members of the visiting staff should be encouraged to take a much greater interest in pathology than they have in the past, for without their active cooperation little of permanent value can be accomplished. Furthermore, bodies such as the American College of Surgeons and the American College of Physicians might well demand of aspirants for admission to their ranks not merely clinical but clinical-pathological reports on their patients. Nor must it be forgotten that our chief aim in this training is the development of a point of view rather than the mere inculcation of facts; for, as Osler well said, "the mental attitude controls the course of a man's evolution as a clinical physician."

195 Church Street.

CHANGES IN INTERNAL MEDICINE SINCE 1900

JAMES B. HERRICK, M.D.
CHICAGO

A fairly complete catalogue of the changes that have taken place in internal medicine since 1900, a catalogue especially adapted to an American audience, could be made if one consulted as source books the *Transactions of the Association of American Physicians,* or the *Index Medicus* and the *Cumulative Index,* or the successive editions of such textbooks as those of Strümpell or of Osler. In fact, this was recently done by Dr. James G. Carr in a scholarly paper entitled "Eleven Editions of Osler." It would, however, be too time consuming for the compiler and too wearisome for the listener if more than the most important changes were discussed, and especially as I shall take the liberty of extending the scope of the topic, so that it will include the last few years of the nineteenth century, for many of the advances in internal medicine of the past thirty-five years were but the continuance of changes that began several years before. I shall also take the liberty of indulging in personal reminiscence. Need we be bound by the dictum of the psalmist that if years are added to the traditional three score and ten they must be years of labor and sorrow? May not one at that age have the privilege and pleasure of going in the direction of least resistance, which is—is it not—toward reminiscence? May not one be permitted to call to mind events that one has seen, if there is avoided the pathetic error of claiming *quorum pars magna fui?*

IMPROVEMENTS IN MEDICAL SCHOOLS AND HOSPITALS

In 1888 I was graduated from a two-year short term medical college, in which the teaching was almost entirely by repetitional didactic lectures. There was no ward work. In anatomy and practical medical chemistry there were laboratory exercises; none whatever in physiology, pharmacology or pathology. I had never seen a case of labor, nor had I myself cut and stained a section for microscopic examination.

However, change was imminent. Thomas Huxley at the opening of Johns Hopkins University School of Medicine had glorified the application of the scientific method to the study and practice of medicine. Young doctors were returning from Vienna and other European medical centers, praising the refinements of clinical diagnosis with impartial postmortem check, speaking

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familiarly of bacteria as a cause of disease, showing enthusiasm over experiment and research. Then Abraham Flexner extolled German methods and advocated having as instructors in medicine those whose life work was teaching and investigation. The Council on Medical Education and Hospitals of the American Medical Association was formed, inferior colleges were weeded out, the standards of others were raised. Medical schools were united to universities. It was declared that a major function of the hospital was to further medical education; internships became obligatory. Richly endowed institutes were founded, entirely devoted to medical research.

Surely one of the significant changes of the last fifty years has been the improvement in medical schools and hospitals and the rise of productive research. The men who teach and those who practice are not necessarily of larger caliber than their forefathers, nor are all the methods in vogue today to be regarded as above criticism or beyond improvement. But, judged by ideals and by results, schools, hospitals and the practice of medicine are of distinctly higher grade.

BACTERIOLOGY

A second major change was the continuing development of the still youthful bacteriology, its application in practice and the outgrowth of such subjects as serology, immunology and allergy.

The germ theory of disease was somewhat slow in getting under way in this country. At the competitive examination for interns at the Cook County Hospital in 1888, William T. Belfield examined in pathology. He had just returned from Europe, perhaps a little puffed up by having been invited to give the Cartwright Lecture in Bacteriology at Yale and at having been asked by William H. Welch regarding some of the technic of staining the tubercle bacillus. I recall most vividly one scene of the examination: The twenty-two competitors, of whom I was one, suddenly stop their writing, gaze blankly and open mouthed as this question is written on the blackboard: "Describe the bacillus of cholera and tell how you would distinguish between it and the Finkler-Prior bacillus." Consternation is on every face. Not a man but knows his mark on that question will be zero. After the examination one candidate, a little bolder than the rest, ventured to protest at such a question on the ground that no college in Chicago, perhaps none in the country, was teaching this subject. Dr. Belfield sarcastically replied that he was well aware of the fact, but that the colleges should be teaching it, and for that reason he had asked the question.

Those who are younger cannot understand the thrill that came as in rapid succession there were announced as fresh discoveries many facts that today are accepted as proved beyond dispute—the germs of suppuration, the antitoxin of diphtheria, the Widal test for typhoid, the transmission of malaria by the mosquito. And the joy that came from identifying for oneself the tubercle bacillus, or from making lumbar puncture and demonstrating the intracellular diplococcus of Weichselbaum! The fun of watching the dance of the pigment granules in the plasmodium of malaria! The miracle unfolded before one's eyes as one injected the 10 cc. of Behring's 1,000 units of antitoxin and watched results! That one first case was proof positive. What about controls? There had been scores of controls in the cases formerly treated empirically with always the spreading membrane, always the fetor, the enlarged glands, the anemia, the fever, the slow convalescence; often the paralysis; or the all too frequent death from exhaustion, laryngeal suffocation or sudden collapse.

One thrill I missed. One evening in 1905 I planned to attend a meeting of the Berlin Medical Society. A German fellow laboratory student advised against it. The program, he said, would be dull. The main paper would be just another unfounded claim of having discovered the cause of syphilis. By those who knew, the writer's contention was practically discredited in advance. I have always regretted that I took my German friend's advice. By so doing I missed hearing Schaudinn announce the discovery of the spirochete of syphilis.

It was not alone that we learned of the causative organisms of so many diseases; not alone that we were aided in diagnosis by identifying these organisms in smears from the throat or in other exudates or even in the blood, or that we were helped by specific blood tests, such as those for typhoid fever or syphilis. It meant much more; for there was unfolded a new conception of infectious disease. A Frenchman once, in writing of malignant endocarditis, said that the disease could be understood if one kept in mind what Virchow had taught concerning embolism and what Pasteur had taught concerning bacteria, and if one looked on the valvular lesion not alone as infectious but also as infecting. This, expressed with Gallic clarity, is what we began to understand — bacteremia with attending toxemia; the transport of germs to distant regions, thus explaining the formerly mysterious and accidental complications; primary and secondary foci of acute infection. And later the misused, much abused, yet fruitful conception of chronic focal infection.

With knowledge of the cause of infections, preventive and active treatment was put on a scientific basis with astonishing results. For years, every summer and autumn, typhoid patients were daily entrants into our wards. One in ten went to the morgue. Today the case in the ward or the morgue is excitedly utilized as a rare educational exhibit of a disease that has almost vanished. Malaria, diphtheria, tetanus, yellow fever, typhus, epidemic meningitis, scarlet fever, uncinariasis, certain types of pneumonia are in the category of diseases for which there is a reasonably reliable preventive or active treatment. "Rats, Lice and History" a generation ago would not, even with the facile pen of its gifted author, have been a title to conjure with. Today it is a best seller to physicians and laymen.

When the true definitive history of the world is written such events as these will bulk large — larger than the story of dynasties and wars. Perhaps they will lead to the use — the horrible use — of bacterial weapons that will prevent future war by revealing its unspeakable and unendurable realities.

INSTRUMENTS OF PRECISION

A third notable change was the invention of many instruments useful in diagnosis and treatment, often spoken of as instruments of precision.

About 1898, at a crowded meeting of the Chicago Medical Society, there were shown for the first time in that city prints made from x-ray plates—the bones of the hand, a few coins and keys in a pocketbook. So crude were these prints that many hearers were skeptical as to the importance to medicine of the discovery of the Würzburg physicist three years before. Two speakers, however, predicted great things, one reminding the audience that modern photography started from

small beginnings by Daguerre, the other stating his belief that some day even stones in the kidney or gallbladder might be shown. How the x-rays have advanced beyond the wildest dreams of those days is familiar to all. Yet it is not always realized how far reaching has been their influence on medicine. They have revolutionized diagnosis and opened up a new type of treatment. Many diseases are incompletely studied if they are not investigated by the x-rays, which often show with marvelous detail pathologic changes otherwise overlooked. Treatment by x-rays as well as by ultraviolet rays and radium is rapidly being established on a scientific basis and gives rich promise for the future.

Numerous other inventions have made some physicians fearful lest the detection of disease would become, in a literal sense, a mechanical process with no place for history, physical diagnosis or logical reasoning, just a robot performance. Not only has the electrocardiograph cleared up many problems as to the heart's normal and abnormal processes; it has become an instrument of practical clinical value. The cavities of the body are illuminated by electric light—by the cystoscope, bronchoscope and other instruments. Blood pressure and basal metabolic rates are determined instrumentally with reasonable accuracy. Local anesthesia makes biopsy or exploratory operation a comparatively simple procedure. One may even class the rubber glove as a device of precision, as it encourages digital examinations that formerly were often omitted because repellent alike to physician and patient. And it is no exaggeration to place among helpful instruments the humble needle and syringe. Exploratory aspiration to determine the presence or nature of fluid accumulations and lumbar puncture for diagnosis or treatment are everyday occurrences. And what a change has been wrought in laboratory and bedside work by the simple withdrawal of blood from the vein, an operation generally delegated to the intern or a technician. The blood is studied for bacteria, for its chemical content, for its physical properties, for its suitableness for transfusions; or remedial agents are injected directly into the blood stream.

CHEMISTRY

Applied chemistry is today indispensable in diagnosis. The order to the intern to "do a blood chemistry" may not be grammatical but it means that the next morning the attending physician knows the amount of blood sugar or of creatinine, calcium or phosphorus. And this order is issued not merely to satisfy an academic curiosity; it is realized that it may disclose diabetes, uremia or hyperparathyroidism.

DEFICIENCY DISEASES

In the nineties we knew of myxedema. I recall the glow of pleasure with which I recognized my first case of cretinism and saw the response to treatment. How keyed up I was when, showing the patient, I devoted an alumni clinic to the topic of the thyroid gland, contrasting myxedema and exophthalmic goiter! Today deficiency diseases are commonplaces. There has arisen the specialty of endocrinology. Closely related and overlapping in borderland territory are certain nutritional and dietary subjects. There is an abundant literature revealing the interest in, and investigation of, such subjects as the underactivity, overactivity or perverted activity of such glands as the thyroid, the adrenal, the parathyroid, the gonads, the pituitary, the pancreas. Pellagra, rickets, beriberi, anemia, diabetes, obesity are understood and treated as never before. Even the laity talks calories, vitamins, hormones, insulin, liver therapy.

OTHER DISEASES

One might go on and cite later knowledge concerning other infections—undulant fever, tularemia, amebiasis, psittacosis. One might refer to the new conception of renal disease; the better understanding of cardiac arrhythmias, the various types of endocarditis, disorders of the coronary arteries, neurocirculatory asthenia; ulcer of the duodenum, diverticulitis—partly revealed by the surgeons; tumors of the lung and bronchi; the more accurate diagnosis and more effective treatment of tuberculosis and of syphilis; new phases of nervous diseases, encephalitis, the stress on psychiatry; the astonishing saving of infant life by the pediatricians; physical therapy; the more intelligent use of tested drugs, e. g., digitalis, arsphenamine, salyrgan, and the lessened use of exploited "patent" remedies. The story of diabetes might be rehearsed with the shifts in treatment through starvation and calculated diets up to insulin, with resulting prolongation of life, or one might tell of the toxic thyroid gland with the brilliant triumph of partial thyroidectomy, the material help from iodine. Those of us who in the earlier days after laboriously translating Ehrlich were fascinated by morphologic studies of the blood, who have seen chlorosis disappear and leukocytosis regarded as a valuable symptom, again have the pulse quicken as we see the results of liver therapy, still eagerly await revelations as to the true nature and the rational treatment of agranulocytosis.

PUBLIC HEALTH

The present generation has seen increased interest in public health. There is a more intelligent attitude toward checking the development and spread of contagious diseases by inspection of schools for such diseases as incipient diphtheria, scarlet fever, the common cold or sore throat. Supplies of water, milk and other foods are more carefully watched. Industrial hazards are diminished by law. Sanitation has become a department of medicine as well as of engineering.

RELATION OF MEDICINE TO OTHER SCIENCES

Another tendency is evident. As no man liveth to himself alone, so no science lives isolated and apart. Medicine, largely through her closeness to other departments of the university, has more intimate contacts with other sciences—physics, chemistry, biology. The results have been mutually beneficial. Physiologists cooperate with chemists, pharmacologists ask help of physicists and botanists, anatomists consort with zoologists and veterinarians; and clinical medicine is the gainer. Furthermore, medicine shows more concern than formerly to regard the patient as a human being; medicine is rubbing elbows with sociology. Perhaps some will say this is not an advance, it is a throwback to the practice of the old time family doctor. At any rate, physicians and the public are considering, more seriously than ever, questions as to the social and economic relations of the doctor, the patient and the state. Health and old age insurance, group practice, qualifications for specialists, rights of physicians as individuals are topics very much alive today.

THE FUTURE

In reading medical history, every once in a while one comes across statements made by men of prominence that little more could be added to the sum of

medical knowledge as it existed at the periods in which these men were living. Only recently I saw practically the same sentiment expressed regarding the present. But I must enter an objection. *Laudatores temporis acti?* Indeed, yes. But not to the disparagement of the present or of the future.

In 1902 at a meeting of the Section on Medicine of the American Medical Association — there were less than 100 in the room—I happened to sit next to Dr. Osler. The new side-chain theory of Ehrlich was being discussed. It seemed to be epochal; it was evidently obscure to Dr. Osler, as it certainly was to me. Seriously, wistfully, Dr. Osler whispered to me, "I wish I were 19 and had it all to do over again." Some of us of the passing generation may wish that we were young so that we might take part in the advances of the future, the solving of the riddles of cancer, endocrines, infections yet untamed or even unnamed, degenerative vascular disease. But with an *ave et vale* we may retire content, assured that internal medicine of the future in its research, as in its practice, will be in safe hands; knowing that when another generation has passed and there is held the seventieth anniversary of the Section on Pathology and Physiology, glorious achievements of the preceding generation will be recounted and that then, as now, a glorious future will be predicted.

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THE DEVELOPMENT OF PATHOLOGY SINCE 1900

WITH SPECIAL REFERENCE TO NEOPLASTIC DISEASES

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Since the beginning of this century the advances in special fields of study have been more marked than in general pathology. Notable among these has been the developing knowledge regarding tumor growth and neoplastic diseases, and it is to that phase of pathology that this presentation will confine itself.

At the close of the last century pathologists were largely engaged with the detailed study of the morphology of tumors, their histogenesis and their separation into different varieties as well as the collation of such facts as pertained to the natural history of malignant disease.

The twentieth century opened with an experimental era of investigation and systematic study of tumors throughout the entire animal kingdom that has brought forth a new conception of various neoplastic diseases and promises to have a profound influence on prognosis and a more successful prevention and therapeutic control.

An epoch making demonstration at the beginning of the century by Jensen [1] in Copenhagen and Leo Loeb [2] in Chicago was that carcinoma and sarcoma could be transplanted and retransplanted in mice and rats for an apparently unlimited time. After more than thirty-four years the Jensen mouse cancer is still being transplanted, and the cells now growing in laboratory mice are descendants of the original Jensen cancer mouse. This suggests that cancer cells are immortal, as are all cells if they can be freed from the limitations of life of the organism as a whole.

These contributions demonstrate an analogy between spontaneous cancer and carcinoma as it occurs in man in two directions: that cancer may arise (1) as the result of chronic irritation of tissues and (2) because of inherited characteristics.

HEREDITY

A number of the early observers, as Leo Loeb,[3] Tysser [4] and Murray,[5] suggested that heredity might play a rôle in the occurrence of spontaneous tumors in animals. It was Maud Slye who first showed on a large scale with mice of known ancestry how important a part heredity plays in producing tumors. This work of Miss Slye has established the fact that, by proper breeding, strains of mice and rats may be obtained with a high incidence of tumors, even independent of the application of any unusual stimulus or other factor to cause tumors to appear. Likewise, the intensive study given to these growths in mice has evidently removed all doubts that they represent fundamentally the same disease of cancer in animals as in man.

The cells of malignant animal tumors resemble fundamentally those of human cancers in that they are embryonal in character, showing mitoses of cells and atypical arrangement, with the tendency to infiltrate adjacent tissues and to invade the blood and lymph vessels, producing tumor emboli and metastases in remote organs.

Wells,[6] who was associated with Maud Slye in a number of her investigations, records having observed in Miss Slye's mice most of the forms of tumors seen in man, even such rare tumors as papilloma arising from the ependyma of the lateral ventricle, malignant adenomatous growth of the anterior lobe of the hypophysis, and primary liver cell carcinoma.

Benign tumors have the same histologic characters and behave as such. Uterine fibroids, teratoma of the ovary, hypernephroma of the kidney and thyroid tumors are alike in mice, dogs and man.

The striking fact has been noted that gliomas of the brain, so common in man, are quite unknown in other species.

These studies in comparative pathology support the view that the inherited constitutional make up of species is an important factor in determining what sort of tumor will occur in that species. A human analogy is furnished by the markedly greater tendency of Negroes to develop fibroid tumors of the uterus and keloid growths than white persons.

The further studies of Maud Slye [7] in comparative pathology promise to throw light also on leukemia, pseudoleukemia and related conditions, which are widely distributed throughout the animal kingdom and seem to correspond closely to similar diseases in man.

In Miss Slye's experience, chronic leukemia and true pseudoleukemia behave like malignant neoplasms, shading off into local lymphosarcoma with metastases. This is in agreement with pathologists who regard these conditions in man as true neoplastic processes.

Whether or not Miss Slye has completely established the heredity mechanism of cancer as a simple mendelian inheritance, as her observations indicate, she has

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1. Jensen, C. B.: Centralbl. f. Bakt. **34**: 28, 1902.
2. Loeb, Leo: Centralbl. f. Bakt. **37**: 235, 1904.
3. Loeb, Leo: J. Cancer Research **6**: 197 (July) 1921.
4. Tysser, C. C.: J. M. Research **12**: 199, 1907.
5. Murray, J. A.: Report of the Imperial Cancer Research Fund, 1911, p. 114.
6. Wells, H. G.: J. M. Research **33**: 171, 1915.
7. Slye, Maud: Am. J. Cancer **15**: 3 (July) 1931.

unquestionably shown more conclusively than was previously done "that heredity is an essentially important factor in determining whether cancer will or will not appear under ordinary conditions of life."

Although the exact mechanism of hereditary influence has yet to be determined, it is safe to maintain the existence of a hereditary influence as to susceptibility and resistance to cancer established both for man and for animals.

There is at present no similar human material to compare with that of Miss Slye in which the complete ancestry for many generations is known. The evidence collected from human material is rather conflicting, being also inadequate as to amount and character.

Wells [8] considers it reasonable to look forward to finding eventually a definite explanation of the genetic mechanism determining susceptibility and resistance to human cancer.

TERMINOLOGY

According to Ewing,[9] the terminology of tumors should designate their etiology, regional location or histologic structure, and of these the histogenesis is the preferable since it suggests the type of cell or cells of a growth and probably serves best by indicating the chief characteristics of its component elements. The histogenic method of classification has specially influenced a newer conception of the tumors of the central nervous and reticulo-endothelial systems.

CENTRAL NERVOUS SYSTEM

A completely new nomenclature has been developed in connection with tumors of the central nervous system. This is based entirely on histogenesis, and the advance of knowledge in this direction is due largely to the fundamental histologic technic developed by Cajal,[10] and Hortego [11] of the Spanish group, and adopted by Bailey,[12] Cushing,[13] Penfield [14] and other investigators in this country.

Two general classes of benign tumors are recognized: meningeal and perineural fibroblastomas. They arise not from within the nervous system itself but from the specialized coverings that separate and insulate the nervous tissue from the rest of the body. The meningeal fibroblastic tumors have their point of attachment from the dura mater and do not invade the brain or spinal cord. In this group are now included psammoma, dural sarcoma, meningioma, endothelioma and arachnoid fibroblastoma.

Under perineural fibroblastomas are grouped neurofibroma, neurinoma, gliofibrosarcoma and cerebellopontile angle tumors. The multiple neuromas constituting von Recklinghausen's disease are also included.

The newer classification of the gliomas, which form from 40 to 50 per cent of all brain tumors, presents a bewildering variety of terms used in their differentiation. To those who are not constantly working with these tissues it may be somewhat difficult to understand clearly all their phases.

Mallory [15] in 1905 showed that a separation of gliomas from other tumors could be made on the basis of cell differentiation and pointed out the distinguishing features of neuroglia fibers.

Bailey and Cushing [16] in 1926, after studying the unrivaled pathologic collection of Cushing's clinic, subdivided 254 verified gliomas on a developmental and structural basis into fifteen classes.

The following year Bailey [17] published a histologic atlas illustrating the different types of gliomas made from preparations stained with the ordinary methods used in pathologic laboratories. The significant statement is made that no two gliomas look just alike, because the gliomas do not fall into distinct groups in which all the members look alike but consist of variant individuals with family resemblances. "It would appear that to find a typical member of each family with which the others may be recognized on comparison is about as difficult as to find a typical member of the Alpine or Dinaric race."

Penfield [18] in 1928 proposed a differentiation into eight classes, adding the statement that groups of tumors such as gliomas may be recognized as histologic entities and their biologic behavior predicted with a considerable degree of certainty.

Roussy and Oberling [19] in 1931, speaking to some extent for the French school, published a classification with an atlas, which is very similar to the one outlined by Penfield, except that the term neurospongioma is substituted for medulloblastoma. Bailey in a later publication, 1932, dropped out certain subdivisions proposed in the original classification, so that the nomenclature is becoming somewhat more simplified.

A knowledge of the histogenesis of the central nervous system is essential for a clear understanding of the tumors of the brain substance known as gliomas. Fundamental is the medullary epithelium with its derivatives on the one hand of spongioblasts, bipolar and unipolar, which grow gradually into astroblasts and astrocytes, and in another direction into ependymal cells, and again to medulloblasts with the derivatives of oligodendroglia and various polar neuroblasts to the final neuron formation.

Further derivatives are the pineal parenchyma and choroidal epithelium. The gliomas are divided generally according to the nature of cells that predominate, comprising three main varieties, as astrocytomas, which are formed essentially by astrocyte elements, the oligodendrocytomas, formed by a profuse multiplication of the oligodendroglia elements, and the glioblastomas, which are formed by young cells of indifferent character resembling more or less the glioblasts of the embryonic nervous system (Roussy).

The classification of Penfield, with the Bailey and Penfield modification, seems to be generally acceptable and includes the main varieties — glioblastoma multiforme, medulloblastoma, astrocytoma, ependymoma, astroblastoma, spongioblastoma, oligodendroblastoma and neuro-epithelioma.

According to Penfield there are three important groups, which together compose three fourths of all these tumors: astrocytoma, glioblastoma multiforme and medulloblastoma. The latter is chiefly a tumor of the cerebellum of infants and young children. Glioblastoma multiforme is a tumor of middle life, its site

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9. Ewing, James: Neoplastic Diseases, ed. 1, Philadelphia, W. B. Saunders Company, 1915.
10. Cajal, S. R.: Paris, A. Maloine et fils, 1909-1911.
11. Hortego, Pio del Rio: Arch. d. cardiol. y. hemat. **2**: 161, 1921.
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13. Cushing, Harvey: Cameron Lecture, New York, Oxford University Press, 1925.
14. Penfield, Wilder: The Classifications of Gliomas and Neuroglia Cell Types, Arch. Neurol. & Psychiat. **26**: 745 (Oct.) 1931.
15. Mallory, F. B.: J. M. Research **13**: 113, 1904-1905.
16. Bailey, Percival, and Cushing, Harvey: A Classification of the Tumors of the Glioma Group, Philadelphia, J. B. Lippincott Company, 1926.
17. Bailey, Percival: Histologic Atlas of Gliomas, Arch. Path. & Lab. Med. **4**: 871 (Dec.) 1927.
18. Penfield, Wilder: Principles and Pathology of Neuro-Surgery, report 15, McGill University, 1932.
19. Roussy, G., and Oberling, C.: Tumors of the Central Nervous System, Arch. Neurol. & Psychiat. **27**: 1281 (June) 1932.

of election being deep in the cerebral hemisphere. The astrocytomas appear in the hemispheres of the cerebrum and in the cerebellum, cerebellar astrocytomas being rarely seen in adults.

The rarer forms of glioma are the remaining more or less clearly defined tumors, as the spongioblastoma, astroblastoma, oligodendroblastoma, ependymoma, neuro-epithelioma and pinealoma. Of these the neuroepitheliomas are the rarest, the most malignant and the least differentiated.

The astroblastomas and oligodendroblastomas are formed chiefly in the cerebral hemispheres. Ependymomas appear near the ventricular walls and the central canal of the spinal cord or filum terminale, while the polar spongioblastomas like the astrocytomas may be encountered anywhere in the cerebrospinal system.

The metastasis of gliomas through the blood stream is almost unknown, according to Davis.[20]

Other types of tumors of the nervous system have been occasionally noted, as ganglioneuromas, gangliomas, papilloma of the choroid plexus, hemangioma, cholesteatoma and chordoma.

The triumphs of neurosurgery are intimately linked with the constantly developing knowledge of the neoplasms involving the central nervous structures.

RETICULO-ENDOTHELIAL SYSTEM

It has been twenty years since the first publications of Aschoff and Landau[21] and of Kiyon[22] proposing "to group together a special type of cells of wide distribution in the mammalian organism as a system of reticulo-endothelial cells" and now generally referred to as the reticulo-endothelial system.

Since then many articles have appeared, particularly as to its rôle in pathologic conditions. In American literature Jaffé,[23] Rinehart,[24] Ewing,[25] Krumbhaar,[26] Goldzieher and Hirschhorn,[27] Caldwell[28] and Giffin[29] have contributed to our knowledge of this phase of pathology.

The reticulo-endothelium has been related to blood formation, particularly the lymphocytes and monocytes, and a third type of leukemia known as monocytic leukemia has been accepted.

Hodgkin's disease is also regarded as belonging to the reticulo-endothelioses.

Ewing[30] states that in the origin of lymphosarcoma two specific cells participate, giving rise to two specific forms of tumor. These cells are (1) the reticulum cell of the germinal centers of the follicles and pulp cords, and (2) the lymphocyte. Thus arise two types of lymphosarcoma, which may be designated as (1) reticulum cell sarcoma, or round cell sarcoma, and (2) malignant lymphocytoma.

When the relationship of the reticulum cells of the mature lymphocytes has been definitely established, it may then be apparent that the reticulo-endothelial system is thus indirectly the source of the malignant lymphocytomas. In that case, lymphatic leukemia and the lymphoid type of aleukemic leukemia may likewise hold a relationship to the tumor-like growths of reticulum cells.

Endotheliomas of the reticulo-endothelial system or reticulo-endotheliomas offer further diagnostic difficulties to the pathologist. More than twenty years ago Ewing emphasized the difficulty of differentiating these endotheliomas of lymph nodes from metastatic carcinoma, especially when the cervical lymph nodes were involved. In more recent years Ewing[31] and Cutler[32] have published rather pessimistic reports concerning the recognition of endotheliomas of cervical lymph nodes because of their close similarity with metastases of transitional cell carcinomas.

Giffin and Watkins[33] have made a distinction between splenic anemia and subleukemia or splenic reticulo-endotheliosis, reporting several cases as leukemic reticulo-endotheliosis.

Rous sarcoma of the chicken is composed of monocytes, but definite tumor formations consisting of monocytes have so far not been reported in man. Tumors originating from the fixed reticulo-endothelial cells are represented by the hemangio-endotheliomas of the liver described by Fischer,[34] Kahle,[35] Goedel[36] and others.

The extent and importance of the reticulo-endothelial system is not generally appreciated, because its physiology and pathology are still rather confusing. It is probable, however, that the studies of hematologists and tissue morphologists will lead the way to a clearer conception of clinical manifestations and their relation to pathologic changes.

HORMONIC ACTION OF NEOPLASMS

The newer revelations regarding the physiologic significance of tumors have tended to modify the pathologic point of view of tumors, particularly of the endocrine organs, and to disprove the older concepts that tumor cells are purely parasitic and nonfunctional.

These observations present a new phase of endocrinology and deal with the possible hormone effects of certain gonadal tumors, more particularly of the ovary and also of the pituitary, parathyroid and adrenal cortex.

Novak and Long[37] presented an interesting discussion before this section two years ago on ovarian tumors associated with secondary sex changes. These neoplasms were of two classes, (*a*) granulosa cell tumors and (*b*) arrhenoblastomas. The former arise from the early oophorogenic structures in the sex gland area, and the histogenesis of the latter, according to Meyer,[38] was to be sought in certain undifferentiated cells persisting in the rete and capable of later function along either male or female lines.

The granulosa cell is a typically feminine cell, producing the ovarian follicular hormone. The hormone effects produced by tumors of this variety are along the line of feminization, with overaccentuation of certain female sex characters and functions.

The arrhenoblastomas, or second group of tumors under consideration, produce effects on the sex characters along quite opposite lines, in that they tend toward a defeminization and masculinization of the individual. Pathologically these tumors are classed as testicular adenomas.

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26. Krumbhaar, E. B.: Internat. Clinics **2:** 280 (June) 1925.
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28. Caldwell, G. T.: South. M. J. **27:** 205 (March) 1934.
29. Giffin, H. Z., and Holloway, J. K.: Am. J. M. Sc. **170:** 186 (Aug.) 1925.
30. Ewing, James: Surg., Gynec. & Obst. **22:** 461, 1916.
31. Ewing, James: Am. J. Path. **5:** 99 (March) 1929.
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34. Fischer, B.: Ztschr. f. Path. **12:** 399, 1913.
35. Kahle, H.: Virchows Arch. f. path. Anat. **226:** 44, 1919.
36. Goedel, A.: Ztschr. f. Path. **29:** 375, 1923.
37. Novak, Emil, and Long, J. H.: Ovarian Tumors Associated with Secondary Sex Changes, J. A. M. A. **101:** 1057 (Sept. 30) 1933.
38. Meyer, Robert: Am. J. Obst. & Gynec. **22:** 697 (Nov.) 1931.

According to Fischel,[39] the close contiguity of the adrenal anlage with that of the gonad, and the fact that both are derived, according to the new view, from the wolffian mesenchyma, make it easy to understand why cortical tumors of the adrenal may give rise to a syndrome very similar to that produced by the tumors originating from the rete region of the ovary. Removal of these tumors has usually brought about a regression of the clinical symptoms.

Reference should be made to the obvious functional rôle of the cells of the acidophilic pituitary adenomas in the production of acromegaly or gigantism and particularly the interesting syndrome reported by Cushing[40] associated with basophil adenomas of the pituitary body.

Pituitary adenomas, according to Cushing, are of three principal varieties, neutrophil, acidophil and basophil, but no constitutional disorder has been definitely ascribed to the basophil variety.

The polyglandular syndrome, formerly supposed to be of cortico-adrenal origin, is characterized in its full-blown state by acute plethoric adiposity, by genital dystrophy, by osteoporosis and by vascular hypertension. At autopsy in six out of the eight instances observed by Cushing it was associated with a pituitary adenoma and in three of the most carefully studied cases it was definitely shown to be composed of basophil elements, the lesion in one instance having been clinically predicted before its postmortem verification.

A further evidence that tumors may possess function is furnished by the studies in recent years of Ballin,[41] Barr, Bulger and Dixon,[42] Jaffé[43] and Wilder[44] or by parathyroidism, in which adenomas of the parathyroid are definitely related to multiple cystic and giant cell tumors of the bone, this evidently being one of the expressions of exaggerated parathyroid activity. When the parathyroid adenomas are removed, a regression takes place in the bone tumors.

Paik[45] has also reported some experiments that show a close relationship between the growth of rat carcinoma and the internal secretion of the parathyroid, the secretion stimulating the development of rat carcinoma, but a decrease of the internal secretion interfering with its development.

GRADING OF MALIGNANCY

Pathologists for some time have considered the possibility of detecting a clinical malignant growth by microscopic examination. In recent years careful studies of carcinoma cells have developed a more concrete knowledge.

About ten years ago Broders[46] proposed a method of grading the malignancy of carcinoma, which has been extensively adopted. It is based on the original studies of Hansmann[47] on the cytogenesis of carcinoma cells and pertains to a dedifferentiation of the cells, a so-called anaplasia or backward formation of epithelial cells undergoing malignant transformation. The grading method of Broders is made on a basis of one to four degrees of malignancy independent of the clinical history. The mitotic figures and one-eyed cells are undifferentiated cells, and in the proportion that the undifferentiated cells appear in the microscopic section as compared to the differentiated cells 25, 50, 75 or 100 per cent the grades of malignancy are stated as 1, 2, 3 and 4.

The confirmation of this method by clinical experience has justified the conclusions on which it is based, and the extensive study of the cellular and histologic structure of carcinoma has added to our knowledge of these neoplastic diseases.

RADIOSENSITIVENESS OF TUMOR CELLS

Within the past twenty-five or thirty years numerous and extensive experiments on animals and abundant clinical evidence have established the fact that every variety of cell in the body, as well as every organ or structure composed largely of one variety of cell, has a specific sensitiveness to roentgen and radium rays.

Earlier investigators—Heineke,[48] Warthin,[49] Krause and Ziegler,[50] Regaud and Cremieu,[51] Aubertin and Bordet[52] and others—demonstrated conclusively that the lymphocytes in the spleen, lymph nodes, intestinal lymph follicles, bone marrow, circulating blood and thymus gland were the most sensitive in the body, the organs next to the lymphoid tissues in sensitiveness to radiation being the testes and ovary. The sensitiveness of the genital glands applies particularly to the basal epithelium of the seminal tubules and the ovarian follicles.

According to Desjardins,[53] the factors responsible for such specificity are not yet determined, but the sensitiveness peculiar to each kind of cell appears to be related chiefly to the natural life cycle. Thus the lymphocytes, the metabolic cycle of which among human cells is the shortest, are also the most radiosensitive, and the nerve cells, the life cycle of which is the longest, are also the most resistant to irradiation.

The susceptibility of normal and pathologic cells of the same kind has been shown to be essentially the same.

More recently Desjardins[54] has proposed irradiation as a means of differentiating certain varieties of tumor and made many other interesting contributions to the subject. His diagnostic classification recognizes three groups as radiosensitive, moderately radiosensitive and radioresistant tumors. In the first group are included lymphosarcoma, Hodgkin's disease, lympho-epithelioma, embryonal carcinoma of the testis and ovary, giant cell tumor and multiple myeloma of bone, basal epithelioma and hemangioma, the moderately radiosensitive growths being carcinoma of the uterine cervix, thyroid, breast and rectum, epitheliomas of tonsils, bronchus, lip, eyelids, mouth, tongue and skin, and chondrosarcoma of bone, while the radioresistant tumors are fibromyoma of the uterus, carcinoma of the esophagus and stomach, mixed tumor of the parotid, hypernephroma, osteogenic sarcoma of bone, melano-epithelioma, teratoma, neurofibroma, lipoma and myoma.

This indicates a comparatively wide variation in radiosensitiveness between tumors which on strictly pathologic grounds have heretofore been regarded as more closely related than they are likely to be in the future.

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The occurrence of malignancy in radioactive persons has been reported by Martland,[55] based on a rather large experience in such cases. Further important papers by Hoffman,[56] Castle and Drinker,[57] Flinn,[58] St. George, Gettler and Muller[59] and Barker and Schlundt[60] covering the nature of the exposure, factory inspections and methods of detection of radioactivity during life with a description of treatment, have added valuable information regarding this new industrial hazard.

Martland's observations were connected with the occurrence of this occupational disease in an accumulative total of about 800 girls employed in a factory in New Jersey, painting the dials of watches and clocks with luminous paint.

The mode of poisoning was by ingestion, arising from a general habit among the workers of pointing their brushes in their mouths while painting the dials, thus swallowing small amounts of radioactive paint day after day.

The following lesions from this mode of poisoning were encountered: irritative hyperplasia of the bone marrow, leukopenias, fatal anemias of the megaloblastic type with leukopenias approaching agranulocytosis, radiation osteitis, necrosis of the jaw, deformities of the spine, spontaneous fractures, multiple myelomas, and osteogenic sarcomas.

Five deaths occurred from osteogenic sarcoma in a total of eighteen cases. The sarcomas evidently developed in areas that had previously been the seat of a radiation osteitis.

It is suggested that radioactivity in the human body can play an important part in the production of other forms of malignancy, as indicated by the high incidence of primary carcinoma of the lungs in the cobalt miners of Schneeberg and in the pitchblende mines of Joachimsthal.

The multiplicity of lesions and diseases produced by irradiation from external exposure to radium and x-rays has been noted by a number of observers.

These observations may have an important bearing on future research on the etiology of cancer and certain obscure blood diseases.

This cursory review of the developments in pathology in thirty-five years might properly be enlarged on in many directions, but the studies recorded in tumor pathology reflect the varied avenues into which pathologic investigations have been extended.

It is significant that pathology has been accorded its proper place among the biologic sciences and withal has maintained its ancillary relation to every other branch of medical progress.

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High Arterial Tension.—There are cases of high arterial tension which are best left untreated. This statement is contrary to much of the generally accepted teaching of the day, but it has been brought home to me forcibly and not infrequently.—Dr. Samuel West, quoted by Fisher, Alexander: Aphorisms in Clinical Medicine, *Canad. J. Med. & Surg.* **77**:166 (June) 1933.

RESULTS AND DANGERS IN THE TREATMENT OF AMEBIASIS

A SUMMARY OF FIFTEEN YEARS' CLINICAL EXPERIENCE AT THE MAYO CLINIC

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An excellent symposium on amebiasis, which gives a summary of the present knowledge of the disease, was presented at the 1934 session of the American Medical Association.[1] This symposium, together with two earlier clinical reports on the results of treatment (Knowles and his associates[2] in India, and Willner[3] in China), suggested reviewing experience at the clinic in the past fifteen years with the type of amebiasis that is encountered chiefly among patients from the north temperate zone.

There have been numerous reports praising or condemning various drugs, pessimistic and optimistic reports on the possibilities of curing the disease, presentations of results of administering drugs based on insufficient experimental or clinical data, and warnings, possibly unduly fearful, of the danger of using different drugs. An outstanding contribution to the chemotherapy of amebiasis has come from the group headed by Leake, Reed and Anderson. It is from such critical studies that not only facts in regard to present drugs but also possibilities of developing new and better ones are obtained. Most workers in this field have long since agreed that emetine in safe amounts fails to cure more than half the cases of amebiasis, and that even doses to the point of toxicity often fail. The organic arsenicals seem definitely established as more effective amebicides, although since arsenic is a well known poison there has been the search to obtain an effective but safe preparation. At present it would seem that there will always be some risk in the use of any effective arsenical because of occasional individual sensitivity. The dose may be safe in most cases, and experiments on animals indicate a wide margin of safety; but it must be remembered that arsenic or any other active drug is a potential poison. One should therefore always have respect for such preparations and use them cautiously.

The oxygenated quinolines have been the third chief type of drugs used in the treatment of amebiasis. Some early reports on chiniofon, introduced as yatren, were most enthusiastic, and in the twelve years in which it has been used its worth has been established. More recently, the administration of vioform has been suggested, as experimentally it was found to be very effective. Too short a time has elapsed, however, to establish its final rating. An even more recent drug for the treatment of amebiasis is diiodohydroxyquinoline, regarding which only a few scattered but encouraging reports are available.

For this period of fifteen years, data on the treatment of amebiasis in 834 cases are obtainable. In 523 of these cases, information as to results of treatment was

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Read before the Section on Gastro-Enterology and Proctology at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 13, 1935.

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secured later. This group of 834 cases comprises a large part of the cases in which a diagnosis of amebiasis has been made in this period. To facilitate analysis of so large a group, the first section of this paper will deal with some of the more commonly used drugs in this fifteen year period, with particular emphasis on reactions; the second section will relate results of treatment.

SECTION I: COMMONLY USED DRUGS AND THEIR REACTIONS

Since ipecac, with its later derivatives emetine hydrochloride, bismuth emetine iodide, auremetine, and the short-lived preparations alcresta, adsorbed emetine and propylcephaline, is the oldest specific drug used in treating amebiasis, it is naturally the one about which much has been written. Ipecac has dropped out of the armamentarium largely because of the great difficulties encountered in maintaining adequate dosage. Of thirty-six patients at the clinic who received ipecac, 50 per cent suffered severe nausea and vomiting so that hospitalization always was necessary (table 1). Even as late as 1927 and 1928, two patients who were resistant to other forms of treatment received ipecac. It seems

TABLE 1.—*Reactions to Drugs*

	Number Treated	Nausea and Vomiting	Neuritis or palsy	Toxic Erythema	Diarrhea	Urticaria	Severe Local Reaction	Theoretical Reactions; Present Dosage, per Cent
Ipecac	36	18*	..	..	..	..	..	....
Bismuth emetine iodide	35	2	..	..	2	..	..	....
Emetine hydrochloride	554	..	16†	..	1	4	2	0.54
Arsphenamine	38	..	..	..	..	..	..	....
Treparsol	301	..	..	8‡	..	..	..	2.6
Acetarsone	232	..	2	13§	..	..	..	....
Chiniofon	37	..	..	..	18*	..	..	....
Vioform	18	..	..	..	..	..	..	....
Carbarsone	8	..	..	1	..	..	..	....

* 50 per cent. † 2.8 per cent. ‡ 2.6 per cent. § 5.6 per cent.

ironical that ipecac at present is remembered only for its pioneer rôle in the treatment of amebiasis.

Bismuth emetine iodide continues to be in some favor, especially in England. It too, however, has the disadvantage of frequently causing nausea and vomiting. In the first seven years of the fifteen, thirty-five patients with amebiasis were given it (table 1); its use was then discontinued, however, in favor of emetine, provided any form of emetine was indicated. Bismuth emetine iodide has the advantage that it can be administered orally, which makes its use advisable in some cases.

Emetine hydrochloride has seemed to be a standby through this entire fifteen-year period. With the advent of treparsol and acetarsone (stovarsol), however, its use decreased somewhat, but it resumed an active rôle in the treatment of amebiasis in conjunction with treparsol. Ever since Sir Leonard Rogers'[4] vivid report (1912) of his first three cases in which emetine hydrochloride was administered, no worker in this field has failed to marvel time and again at one of the therapeutic miracles in medicine: the prompt response of active and acute amebiasis to 0.19 Gm. or 0.26 Gm. (from 3 to 4 grains) of this drug. At the clinic, emetine hydrochloride has always been administered subcutaneously, and it is well to note that much undue local reaction is avoided by observing care in injecting it subcutaneously; it is not injected intramuscularly or intradermally, and we see no need of intravenous administration.

Naturally, with the widespread use of emetine there soon appeared discouraging reports about its failure to effect a cure in more than half of the cases. Occasional reports also appeared of neuritis or palsy, myocardial injury, profound local reactions at the site of injection and, finally, deaths associated with its use. I have been able to find reports of ten deaths attributed to emetine in the period from 1912 to 1935. The United States Bureau of Vital Statistics has no records of death due to emetine poisoning in the United States, although there are two or more reported in the literature. An article by Chopra and Ghosh[5] in 1922 quoted Hall and Dalimier as each reporting three fatal cases. Unfortunately the references were omitted, and I can find only Dalimier's article in 1917,[6] quoting in turn two cases of emetine neuritis or palsy. By no means is there any intention to minimize these tragedies, yet with the millions of injections this small number of untoward reactions is extraordinary. In these ten fatal cases the dose received ranged from one dose of 0.02 Gm., given to a child aged 2 years,[7] who died very suddenly after the injection, to 2.64 Gm. administered in the course of eight weeks. Leibly's[8] case (1.28 Gm. in eight weeks, equivalent to 34 mg. per kilogram) and Levy and Rowntree's[9] case (1.74 Gm. in eighteen days, equivalent to 25 mg. per kilogram) are the only two cases in which I find data as to the dose per kilogram of body weight. The other seven patients received 1.07, 1.44, 1.04, 1.52, 1.08, 1.88 and 0.48 Gm. in periods of from two to eight weeks.

In the series at the clinic of 554 patients who received emetine (table 1) the usual dose ranged from 0.24 to 1.17 Gm., the latter dose being given in a month's time and being not more than 17 mg. per kilogram. In the past seven years the usual dose has been from 0.39 to 0.65 Gm. in any one month, or about 9 mg. per kilogram (estimating an average normal of 70 Kg.). There have been no deaths in these 554 cases except of one patient who was moribund on admission as a result of peritonitis from a ruptured abscess of the liver; death occurred seventy-two hours later. He had received 0.39 Gm. of emetine in that time.

The not tragic but nevertheless distressing complications of emetine therapy are peripheral neuritis or palsy and cardiovascular disturbances. It is well to remember that the effect of emetine is cumulative and that it is eliminated slowly just as is the arsenic in acetarsone and carbarsone. In 1920 Mattei[10] demonstrated this fact and in one case found emetine present in the urine sixty days after an eight day course of 0.48 Gm. of the drug. No doubt not all deaths have been reported, and certainly many of these less serious reactions have not. I have found thirty-seven such cases in the literature in which the average amount of emetine given was 1.0 Gm., eight of the patients having been poisoned by 0.65 Gm. or less. This gives no idea of the incidence of injury to nerves or myo-

4. Rogers, Leonard: The Rapid Cure of Amoebic Dysentery and Hepatitis by Hypodermic Injection of Soluble Salts of Emetine, Brit. M. J. **1**: 1424-1425 (June 22) 1912.

5. Chopra, R. N., and Ghosh, B. N.: The Therapeutics of Emetine, Indian M. Gaz. **57**: 248-253 (July) 1922.

6. Dalimier, R.: La toxicité du chlorhydrate d'émétine, Presse méd. **25**: 33-35 (Jan. 18) 1917.

7. Stern, E.: Trop. Dis. Bull. (abstr.) **7**: 237, 1916.

8. Leibly, F. J.: Fatal Emetine Poisoning, Due to Cumulative Action, in Amoebic Dysentery, Am. J. M. Sc. **179**: 834-839 (June) 1930.

9. Levy, R. L., and Rowntree, L. G.: On the Toxicity of Various Commercial Preparations of Emetine Hydrochloride, Arch. Int. Med. **17**: 420-443 (March) 1916.

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cardium. Only two cases, with accompanying electrocardiograms, have been reported in which it was felt that there was cardiac injury; in one [11] the vagal influence had not been ruled out by atropine and in the other [12] the patient had received digitalis prior to the taking of the electrocardiogram; the tracing was normal three weeks later. There is no question that intoxication is manifested by rapid pulse, asthenia, vomiting and diarrhea and that it often goes on to frank neuritis or palsy. As to how much actual myocardial injury is produced in patients who survive is still debatable. It has been suggested that tachycardia [13] is due more to irritation of the vagus nerves than to the myocardial injury. The experiments on animals [14] leave no doubt that emetine is a protoplasmic poison, while the nerve lesions are the result of degeneration of fibers in the motor roots and not actual neuritis. Clinically, emetine should be used at a point far below toxicity, leaving only a very small chance of reaction in the exceedingly hypersensitive individual. In many of the reported cases of intoxication and in our own there have often been warnings of intolerance which in retrospect are quite definite. In some cases the reaction appeared without any warning.

The probability of myocardial injury increases at the age of 60 years and beyond, so that one might expect to note some evidence of cardiac disturbance on giving emetine to older patients. There are data on twenty-five patients whose ages extend from 60 to 74 years, all of whom received from 0.26 Gm. (4 grains) to 1.1 Gm. (17 grains) in from two to four weeks' time (an average of 0.65 Gm., or 10 grains). In two cases neuritis developed; in one the symptoms came on after 1.1 Gm. (17 grains) of emetine had been given, and the patient suddenly recalled having recently received emetine elsewhere. Throughout the first three weeks during which neuritis was most marked, this patient's blood pressure held constantly at 175 to 180 mm. of mercury systolic and 90 to 94 mm. diastolic, with a pulse rate at 80 beats a minute. The second patient was not seen at the clinic after neuritis appeared, as it had followed a second course of 0.26 Gm. (4 grains) of emetine administered by his local physician. Report was made only of weakness of the extremities. The remaining twenty-three patients showed no intolerance to the drug.

In the 554 cases at the clinic in which emetine was given (table 1), there were eight in which there was generalized asthenia and weakness of the extremities to an extent that would justify classifying it as a neuritis or palsy. In eight other cases, exhaustion and general depletion were definite and possibly should likewise be considered as a mild palsy. The first eight patients received emetine in doses of 1.1, 1.04, 0.65, 0.52, 0.91, 0.97, 0.65 and 0.78 Gm. Six of the second eight patients received 0.78 Gm., one received 0.75 Gm., and one received 2.73 Gm. In no case was there any particular reference to disturbance of the cardiovascular system. In six of these sixteen cases there were early warnings after the first course of from 0.24 to 0.39 Gm. had been given, such as hives, increased diarrhea, joint pain and weakness, but the administration of emetine was continued. In ten cases the symptoms appeared shortly after completion of the course of emetine. One must accept the entire sixteen, or 2.8 per cent of the 554 cases, as the incidence of emetine reaction; with our present average dose of 0.65 Gm., however, it is noted that only three of the sixteen reactions occurred at this dose or less. Merely for contrast, as well as to emphasize the all important factor of individual tolerance, I have the record of three patients who received 8.3 Gm. (125 grains), 8.7 Gm. (134 grains) and 11.6 Gm. (180 grains) respectively in a period of from eight to twelve months; in addition, many others have received two or three times the usual 0.78 Gm. in three months' time. In none of these cases were there signs of intoxication. In two additional cases urticaria was prominent, and in still two others large ecchymotic areas, with induration, developed at the sites of injection. The last two might have been due to errors in technic or perhaps to a faulty product.

From these observations in 554 cases it hardly seems justifiable to discard emetine, although it should be reemphasized that emetine in the total dose of 0.65 Gm. in two weeks is employed only to control acute manifestations of the disease and to give the patient prompt relief but not with the idea of continuing with the drug to bring about a cure. With this in mind, exception will not be taken to my stating that three reactions to 0.65 Gm. or less in 554 cases (0.54 per cent) is about the incidence of reaction or idiosyncrasy to emetine.

Organic Arsenicals.—Arsphenamine, acetarsone and treparsol have been the arsenicals used at the clinic (table 1). In the last two years we have had occasion to employ carbarsone, but in too few instances to justify any conclusions. From the studies of Reed, Anderson, David and Leake,[15] carbarsone is shown to produce fewer reactions than either treparsol or acetarsone, although I have had reported to me two cases of toxic erythema and one case of neuritis. The slow elimination of carbarsone, as with acetarsone, suggested the possibility of an occasional neuritis. Arsphenamine has been put aside not so much from fear of it but rather because the expense and time element favored orally administered drugs. Thirty-eight patients received arsphenamine, and with no untoward reactions.

Acetarsone was thoroughly tried and seemed to be the answer to the problem of an easy, inexpensive drug that would complement emetine, emetine being used for control of acute symptoms and acetarsone for eradication of the amebas. Deaths are recorded in several reports, but in the 232 cases in this study (and in many others in which acetarsone was prescribed) a death has not occurred. Toxic erythema has been noted, however, occasionally being severe enough to be considered as dermatitis exfoliativa; toxic erythema was encountered in thirteen (5.6 per cent) of the 232 cases in which patients received acetarsone (stovarsol). Three patients were quite ill for a week; the others were moderately sick and the symptoms abated in from forty-eight to seventy-two hours. Severe peripheral neuritis occurred in one case, recovery from which took almost a year. A second but milder case occurred, and the patient was well in from six to eight weeks. In both instances arsenic was present in the urine months after the administration of acetarsone had been stopped.

In view of these two cases of neuritis and thirteen of toxic erythema it was realized that the risk of treat-

11. Anderson, H. H., and Reed, A. C.: Untoward Effects of Antiamebic Drugs, Am. J. Trop. Med. **14:** 269-281 (May) 1934.
12. Chopra, R. N., and Sen, B.: Toxic Effects of Emetine on the Cardiovascular System, Indian M. Gaz. **69:** 262-263 (May) 1934.
13. Cawston, F. G.: Emetine Poisoning, J. Trop. Med. **32:** 22-24 (Jan. 15) 1929.
14. Young, W. A., and Tudhope, G. R.: The Pathology of Prolonged Emetine Administration, Tr. Roy. Soc. Trop. Med. & Hyg. **20:** 93-99 (March-May) 1926. Anderson, H. H., and Leake, C. D.: The Oral Toxicity of Emetine Hydrochloride and Certain Related Compounds in Rabbits and Cats, Am. J. Trop. Med. **10:** 249-259 (July) 1930.
15. Reed, A. C.; Anderson, H. H.; David, N. A., and Leake, C. D.: Carbarsone in the Treatment of Amebiasis, J. A. M. A. **98:** 189-194 (Jan. 16) 1932.

ment was considerable and that it was particularly dangerous to prescribe the drug unless the patient was under constant observation.

Flandin [16] stated that the arsenic of treparsol was eliminated within two or three days after completion of a course of this drug. This suggested that the neuritic complication could be avoided and the immediate acute reactions perhaps lowered. Flandin's statement as to the rapidity of elimination was confirmed and, clinically, no cases of neuritis have occurred. Since 1925, 301 patients have received treparsol, and it was prescribed for others whose treatment was carried out elsewhere. There occurred eight, or 2.6 per cent, instances of toxic erythema in the 301 cases. One additional patient had nausea and vomiting after taking four tablets (1 Gm.), and administration of the drug was discontinued rather than risk any chance of reaction. Four of these eight patients manifested erythema at the end of the first course (3 to 3.75 Gm.), whereas four showed reactions during or toward the end of a second course of 3 Gm. Symptoms of all subsided in from three to five days. While deaths from treparsol have been recorded, none have occurred in cases at the clinic.

While chiniofon ("yatren," "anayodin") has been regarded with favor at the clinic and employed since 1926, it has not been the drug of first choice. Possibly

TABLE 2.—*Results Obtained with Specific Drugs*

	"Cure"		Failure	
	Cases	Per Cent	Cases	Per Cent
Emetine	88	55	73	45
Ipecac	11	73	4	27
Arsphenamine	10	91	1	9
Acetarsone	112	85	19	15
Treparsol	34	89	4	11
Chiniofon	22	82	5	18

the earlier item of expense (when it was imported as "yatren"), as well as the frequency with which it temporarily increased the diarrhea, first led to the use of emetine, in conjunction with treparsol. Chiniofon is noted as being used in only thirty-seven of the 834 cases (table 1). This gives a rather unfair picture, however, for most of these patients had received previous, and unsuccessful, treatment with emetine and arsenic. Aside from the rather frequent inconvenience of increased diarrhea on the usual dose of 3 Gm. daily, no untoward difficulties occurred. The two deaths that followed the intravenous administration of chiniofon in cases of actinomycosis are quoted constantly and indicate that the drug is not without some danger. However, the danger would seem minimal in the usual doses administered orally.

Vioform is another halogenated quinoline that has been used at the clinic. As it was available only during 1933 and 1934, it has been tried in only eighteen cases (table 1). There were no difficulties and less irritation from its use than with chiniofon. As with carbarsone, we have had too little experience to offer any conclusions. Recently, a third compound of this group has been offered for trial, diiodohydroxyquinoline. In only one case has it been possible to follow up the patient, and in this case stool tests were negative three months after treatment.

The use of colonic irrigations of any of the foregoing medicaments has, unfortunately perhaps, not been tried. I have not been convinced that they added to the treatment, and no doubt there was an underlying prejudice against colonic irrigations.

As a matter of record, experiences with retention enemas of coal oil should be noted. In most instances there was no difficulty other than the attendant nuisance plus a persisting aroma of kerosene during treatment. A few patients would smell and taste kerosene in eructations of gas, which quite definitely was the result of reverse peristalsis. In one case a severe and rather dramatic reaction occurred. Immediately after receiving coal oil, the patient complained of a very tight feeling in the chest. The respiration rate rose to 36 per minute and the temperature to 102 F. Coarse râles were heard in both lungs. The patient was very restless and apprehensive, and grave anxiety was held as to the outcome. The symptoms slowly subsided, however, and in twenty-four hours he had entirely recovered.

SECTION II: RESULTS OF TREATMENT

In the evaluation of results of treatment it is essential not only to have later and repeated examinations of stools but also to have information as to what types of amebiasis are encountered in any particular report. The effectiveness of treatment in a group of cases of latent amebiasis might be quite different from that in a similar number of cases presenting hepatic abscesses or acute dysentery.

In this review, 278 of the 834 patients (33 per cent) had evidence of active amebiasis on admission, 424 (50 per cent) had intermittent diarrhea associated with various abdominal complaints, some of which were and some of which were not improved by antiamebic treatment, and 132 (16 per cent) were considered as having latent amebiasis. In the latent group were listed fifty-one patients with chronic ulcerative colitis, in all of whom colitis had persisted after antiamebic treatment. There is no doubt that the relative percentage of latent cases can be varied almost at will, depending on how routine the examination of the stool is. Since the stools of patients who come to the clinic are not all examined and, further, as this group of 834 cases consists merely of those in which data as to treatment are available, the foregoing percentages do not give a true ratio of the incidence of the various degrees of amebic infection in the north temperate zone.

Of the total group of 834 cases, 523 afforded sufficient data to warrant tentative conclusions as to the results of treatment. It is realized that in many the ideal of repeated examinations over a prolonged period has not always been possible, yet I believe that the follow-up data have been sufficiently adequate to indicate what has transpired (table 2). The word "cure" is used in this paper to imply that not less than two examinations of stools were made on successive days following completion of treatment, while in most cases repeated examinations were made over a period of months to several years. In addition, clinical symptoms of amebiasis no longer existed. "Failure" implies that the parasite was identified in the stool after treatment was completed even though there were no clinical symptoms. In contrast to institutional patients, our patients had returned to their original environment, so that reinfection might have occurred; yet we have considered as failures any cases in which the parasites were found after conclusion of treatment.

Treatment with emetine consisted usually in giving an average dose of 0.78 Gm. (12 grains) over a period of four weeks. In most cases a series of kerosene enemas were given during the month, and in a number

16. Flandin, C.: Le dérivé formylé de l'acide méta-amino-para-oxyphényl-arsénique (tréparsol) dans le traitement de l'amébiase, Bull. et mém. Soc. méd. d. hôp. de Paris **2**: 1628-1636 (Nov. 21) 1924.

of cases chaparro amargoso was administered orally. Hence whatever virtue there may have been in kerosene and chaparro, they did not enhance the value of emetine as a curative agent to any marked extent. The observation of emetine failing as a curative agent in 45 per cent of cases is in accord with that of many other observers. Ipecac, in the few cases in which it was used, shows a better result, but one that is still far from satisfactory; in addition there is marked unpleasantness from its oral administration in 50 per cent or more of cases. Bismuth emetine iodide was frequently used to supplement other treatment, yet as a primary weapon of attack it seems to have been used in only two cases of which we have record, "curing" the patient in one and failing in the other. Nausea and vomiting and diarrhea occur in some cases, although they are less severe than with ipecac.

TABLE 3.—*Combinations of Emetine and Arsenicals*

	"Cure"	Failure
Emetine and arsphenamine	7	..
Ipecac and arsphenamine	1	..
Emetine and treparsol	111	15
Emetine and acetarsone	13	3
Ultimate results with emetine and arsenic	142*	8†

* 94.7 per cent. † 5.3 per cent.

The primary results of treatment with the organic arsenicals is much better than with emetine or ipecac. Failure to obtain a cure, in addition to the occasional reaction, in only 9 to 15 per cent of cases, is most encouraging although still not at the desired goal.

Too few results are available on the efficacy of the oxygenated quinolines. It is not because of fear or prejudice but rather because there has been such a comfortable feeling with the emetine-arsenic regimen since before the quinolines were available that we have used them later more as "shock troops." In eight cases chiniofon (yatren) was used primarily, and in fourteen cases secondarily, for a total of twenty-two cures. Failure resulted in one case in which treatment was primary and in four cases in which it was secondary. This is not a just estimate of the drug's value, as the eighteen cases in which treatment was secondary indicate that a more stubborn infection existed. Likewise, too few results were available on the efficacy of carbarsone and vioform. In only one case was vioform used alone; in this case active amebic dysentery persisted in spite of the fact that emetine had been administered elsewhere to the point that peripheral neuritis had developed. Vioform failed to result in a cure. In two cases it was combined with treparsol and a cure was effected. In fifteen other cases vioform was given but the results of treatment were unknown. Nine patients received carbarsone, but data are available for only three of them. Used alone, carbarsone failed to cure; in conjunction with chiniofon one patient was cured, and with emetine one failed.

The high percentage of failures with alkaloids and the much better results with the arsenicals naturally suggested the value of combining emetine and an arsenical, the former to be used only in sufficient dosage to control acute symptoms, thereby keeping well below even a minimal danger of poisoning, and the latter to be used in smaller doses and yet prove effective in eradicating the amebas. The results noted in table 3 confirmed this opinion, for in 150 cases there were eighteen primary failures, or 12 per cent, on treatment at the clinic. Ten of these patients were later cured, leaving eight regarding whom no further data were available. The final results were that treatment failed in 5.3 per cent and was successful in 94.7 per cent of the cases.

Results of Treatment After Original Failure.—This is the group that naturally causes the most anxiety of all and serves as the stimulus to more effective treatment. As previously noted, the incidence of the more severe manifestations of amebiasis is probably higher in the tropics, so that any reports from the north temperate zone may not be comparable to those originating in the Orient, in India or in Panama. In the 523 cases at the clinic 398 primary cures were obtained, but it is the 125 failures that are of the most concern (table 4).

Striking improvement coincides with the wider use of the organic arsenicals. It also signifies that the profession has ceased to look to emetine as a curative agent but rather as a means of controlling the acute symptoms. Of the 125 patients whose original treatment failed, sixty were later cured; twelve persisted with the disease in spite of continued treatment, and for fifty-three no further data were available. It may be unfair to include all these fifty-three cases as final failures, just as it is rather optimistic to include all the remainder as true cures, but nevertheless sixty-five are listed as failures. But to consider these sixty-five cases as final and absolute failures is not justified, for as time goes on the majority of these present failures gradually become apparent cures, which observation has repeatedly been confirmed.

With present knowledge, the "reclaiming of failures" lies in a systematic and conservative variation in the treatment. If failure has followed the use of emetine and treparsol, success is unlikely to follow their continued administration; likewise, the possibility of drug reaction increases. Two or three courses of chiniofon are prescribed (21 Gm. a week, with a week's interval between courses). If amebas persist, six weekly injections of arsphenamine or bismuth emetine iodide, with large doses of bismuth subcarbonate, are employed. In other words, varying the ammunition minimizes reactions to the drugs and seems more effective in finally obtaining a cure. As one reviews the sixty cases finally considered as cured, the barrage of arsphenamine, emetine, bismuth emetine iodide, chiniofon, carbarsone, acetarsone, treparsol, vioform and ipecac is indeed startling, and one asks what good any one drug is when such a variety is used. In any event, a cure was eventually obtained.

TABLE 4.—*Results of Original and Later Treatment*

	Total	"Cure"		Failure	
		Cases	Per Cent	Cases	Per Cent
Original treatment					
1920 to 1925	187	111	59.4	76	40.6
1925 to 1935	336	287	85.5	49	14.5
Total	523	398	76.1	125	23.9
Final results					
1920 to 1925	187	152	81.3	35	18.7
1925 to 1935	336	306	91.1	30	8.9
Total	523	458	87.6	65	12.4

Of the twelve cases in which failure persisted even after further treatment, five occurred in 1920 when more emetine and ipecac were given. One patient later received one course (4 Gm.) of acetarsone, but without avail; at present I would not consider that sufficiently varied and persistent treatment had been given.

One patient had a course of six injections of arsphenamine after the course of emetine had failed. Another patient received 1.52 Gm. of emetine (24 grains) on top of an original course of 0.78 Gm. (12 grains). The other five patients received fairly intensive treatment with emetine, acetarsone, treparsol and chiniofon, but in spite of this the parasites were not eradicated. Although these five cases were still failures, yet of the sixty eventual cures in the original 125 failures these sixty had as much or more treatment, some for two to three years. Hence it is hoped that judicious and varied treatment with the present drugs will result successfully in almost all cases of amebiasis encountered at the clinic.

The figure 91.1 per cent as representing cases in which an apparently final cure was obtained in the past decade will undoubtedly provoke criticism, and I realize that, if repeated examinations of the stools of these patients could be made over a period of months, the final result would be less. On the other hand, instituting the program of variation in further treatment makes me optimistic enough to believe that the final outcome would yield as high or even a higher percentage. As one respects the potential dangers of the drugs that are employed, so the recognition of the ameba as a wily and stubborn enemy results in a more determined and judicious management. Again it must be stated that those manifestations of the disease are not encountered when one considers ileostomy or appendicostomy, or colostomy for rectal stricture.

TABLE 5.—*Results of Treatment on Amebic Rectal Ulceration*

	Total	"Cure" Cases	"Cure" Per Cent	Failure Cases	Failure Per Cent	No Data
Amebic proctitis	195	115	82*	26	18*	54
No proctitis	330	174	84*	34	16*	122
Chronic ulcerative colitis	51	36	...	1	...	14

* Percentage based only on cases in which further data were available.

Among the more serious complications of the disease to be dealt with should be noted clinically recognizable injury to the liver, which occurred in twenty-two cases. In sixteen cases there was abscess formation, whereas in six there were chills, fever, leukocytosis, and tenderness in the region of the liver, which certainly suggests hepatic involvement. As has been stated, one of the patients was practically moribund on admission and died of general peritonitis. Drainage of the abscess had been established and 0.39 Gm. (6 grains) of emetine had been administered. Necropsy revealed that an abscess had ruptured into the peritoneal cavity.

In the remaining fifteen cases of known abscess, aspiration or open drainage had been done, accompanied by antiamebic treatment. So far as we know, all these patients have been cured. Five of the six patients with hepatitis likewise have remained well, although the sixth, treated late in 1934, has written that she is still having some trouble.

With the exception of the patient who died and two others who were treated in 1920 and 1924, all patients received neoarsphenamine, acetarsone or treparsol in conjunction with emetine. In no case was there evidence of untoward reaction or of an increase in hepatic injury. In fact, in one case convalescence was prolonged and more complete recovery occurred only after treparsol was given; treparsol had been withheld for fear of injuring the liver. With even nineteen cases of hepatic injury it is not proper to state that the arsenicals are not dangerous, but in cautious and interrupted courses their administration has not only not caused harm but has proved beneficial.

Another aspect of the results of treatment of the more severe types of amebiasis is seen in cases in which amebic rectal ulceration could be demonstrated. Of 576 patients who were examined proctoscopically, 195 (33.8 per cent) had amebic proctitis, 330 (57 per cent) were normal, and 51 (8.8 per cent) had idiopathic ulcerative colitis. The last were cases in which amebas played only an incidental rôle and the colitis persisted after the amebas had been eradicated. Several patients in the first group had some associated stricturing, but only one had a marked tubular stricture. The last, a Nicaraguan, is one of a group of patients who received combined antiamebic treatment and local treatment of the rectum for more than three years; a cure was finally obtained and the patient had an adequately functioning rectum (table 5).

PRESENT METHODS OF TREATMENT

If the patient has not received antiamebic treatment recently, he is given 0.065 Gm. (1 grain) of Burroughs, Wellcome & Co. emetine hydrochloride, subcutaneously, twice daily for three days. After an interval of a week, 0.043 Gm. (two-thirds grain) of emetine is given twice daily for three more days. With the institution of the emetine, treparsol, 0.25 Gm. (4 grains), is administered orally with each meal for four days. If there is no intolerance to arsenic, two more such courses are prescribed with intervals of ten days between the courses.

If the patient is quite ill, he is kept in bed for the first few days; if he is not particularly ill, hospitalization is not necessary. Obviously the diet may need to be bland and simple if there is much dysentery, but very rapidly, that is, within twenty-four to forty-eight hours, a full and generous diet is begun. In any depleting disease, adequate amounts of nourishing, utilizable food are most essential. As recently demonstrated in experimental amebiasis by the Tulane group (Faust, Kagy [17] and others), the importance of a rich, high vitamin diet had a profound influence on the healing of amebic ulceration. Hepatic involvement may subside, but if there is a large collection of broken down material, aspiration preferably, or occasionally open drainage, may be required.

If stool tests are positive following this regimen, three courses of chiniofon are prescribed: 3 Gm. orally per day for a week and repeated for two more such courses, with a week's interval between courses. If diarrhea is increased, the daily dose is decreased, thereby prolonging each course. Failure after this would indicate a course of one injection of arsphenamine weekly for six weeks, and 1 drachm (3.88 Gm.) of bismuth subnitrate from three to six times daily during the period. As it was aptly expressed by Anderson and Reed,[18] "No one drug is known today to be completely effective . . . and the therapeutic hazard should not exceed the disease hazard." Hence there is need to vary the treatment in regard to the individual patient as well as to the type or degree of severity of the infection. With continued search for more effective and safer drugs, and with a better knowledge of the "soil" of the patient, this present regimen will be simplified.

17. Faust, E. C., and Kagy, E. S.: Studies on the Pathology of Amebic Enteritis in Dogs, Am. J. Trop. Med. **14**: 221-233 (May) 1934.
18. Anderson, H. H., and Reed, A. C.: Carbarsone Rectally in Amebiasis, Am. J. Trop. Med. **14**: 257-267 (May) 1934.

SUMMARY AND CONCLUSIONS

1. The use of 0.65 Gm. (10 grains) of emetine hydrochloride subcutaneously in a month's time should involve a risk of reaction in less than 1 per cent of the cases.

2. The use of any of the present organic arsenicals is attended by some risk, but by observing the rule of interrupted courses as well as observation of the patient this risk is minimized.

3. In our experience at the clinic, acetarsone, treparsol and carbarsone have produced reactions. Clinically acetarsone produced the most and carbarsone the least.

4. The use of the arsenicals has not proved detrimental in the treatment of amebic liver abscess.

ABSTRACT OF DISCUSSION

DR. MOSES PAULSON, Baltimore: Dr. Brown has comprehensively reviewed the pharmacotherapeutic measures in amebiasis. I agree with his conclusion that the vast majority of amebiases encountered in the north and midtemperate zones respond completely to the use of one or more of the several measures available. I should like to stress, because of the not infrequent recurrences, that patients should be observed for a longer period of time than is generally done before they are discharged as "cured." Since I have seen recurrences—possibly reinfections—in the fourth month following seemingly adequate therapy, Andrews and I, in work a report of which we are about to publish, state that we believe that a "cure" should not be regarded as having been attained until three fecal specimens monthly, obtained preferably after a saline purge, are negative over a period of six months. Dr. Brown has indicated the relative harmlessness of emetine hydrochloride in amebic dysentery. This must be emphasized, since undue references to its dangers have appeared recently, and there is greater danger that an important therapeutic agent may be discarded. It is striking that in his hands only slightly more than 0.5 per cent of cases manifested untoward reactions. Besides, there is no authentic report of myocardial damage in any adult receiving a total of 0.65 Gm. or less. If there is no satisfactory response to this dose, larger doses are usually of no avail. These are important considerations, for I believe that emetine hydrochloride is the best drug in amebic dysentery. It is a prophylactic as well as a specific in amebic hepatitis. The action of emetine is not understood; it is thought that it attacks the tissue invaders; therefore it must be complemented by another drug—usually an arsenical—to destroy the lumen dwellers. At the Johns Hopkins Hospital, in amebic dysentery, I use emetine subcutaneously, simultaneously with the arsenical carbarsone by mouth. Immediately on cessation of acute manifestations, emetine is discontinued but carbarsone is continued. Recurrences are treated with another course of carbarsone. The rare further recurrence is treated with the oxyquinoline derivative vioform. In carriers I have never given emetine: carbarsone is administered and used for recurrences. In subsequent recurrences vioform is used. Carbarsone has been extremely satisfactory and no untoward reactions have been noted.

DR. SIDNEY SIMON, New Orleans: My experience is that the treatment of this disease, which extends back for more than thirty years, doesn't coincide in a great many particulars with the results that Dr. Brown has reported. Ipecac was the first drug that modern medicine used in the control of amebiasis; emetine was discovered three years after the reintroduction of ipecac and was used almost to the exclusion of ipecac by most men for a period of almost fifteen years. In Dr. Brown's first paper, the only effect of ipecac, the only reaction, as he called it, was nausea and vomiting. There was no other untoward effect. Ipecac can be given in massive doses without any untoward effect other than the nausea and vomiting, which can be controlled to a great extent by careful administration. The ill effects of emetine have been grossly overemphasized; however, emetine is an undesirable drug in amebiasis for the reason that it is inadequate. Emetine is much like morphine, an excellent drug to use in emergencies of acute amebiasis, when one has to control the immediate symptoms of an acute dysenteric attack. Ipecac, on the other hand, in more than 500 cases that I tabulated several years ago, proved completely successful in more than 90 per cent as compared to the rather low percentage that Dr. Brown has given. The treatment of amebiasis should be standardized. The treatment has been passed back and forth as a football. There is a modern method of treating amebiasis and a successful one, and that is that one has the choice of two modern drugs, both of which are eminently successful: carbarsone, preferably, which gives a very high percentage of good results without any toxic arsenical effect, and either vioform or chiniofon, which are quinoline derivatives. The curative effect fails in less than 10 per cent with these drugs. Then there is ipecac to fall back on, which I am still holding in reserve as a means of curing cases that cannot be cured by emetine. Emetine is the worst drug that can be used in the resistant cases; carbarsone or vioform is better. Ipecac is still a drug to be reckoned with as almost a specific in amebiasis.

DR. PHILIP W. BROWN, Rochester, Minn.: The question of what our present regimen of therapy is and what we mean by the word "cure" is covered in detail in the paper. I have tried to avoid talking about vioform. I have used carbarsone rather extensively, but it seems to me that there are differences in what can be accomplished with them. Vioform has proved inefficient in my hands, while carbarsone has proved much safer, but less efficient than treparsol. For these reasons we seem to stick to the drugs that have given us the best results.

THE REDUCTION OF DIPHTHERIA FOLLOWING THREE DOSES OF TOXOID

FURTHER OBSERVATIONS

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In 1926 the Department of Public Health of the City of Toronto offered immunization with diphtheria toxoid to children in the public schools of the city.[1] Such immunization was carried on till June 1929. Advantage was taken of the opportunity to gain an accurate estimate of the efficiency of toxoid in preventing diphtheria in the group so treated. The results of that study, with the methods of calculation, have been published.[2] Table 1 presents, in summary, the data of that time. Here the inadequacy of one dose is at once apparent and needs no comment. The advantage of a secondary stimulus in immunization is evident in the much greater reduction of diphtheria (74 per cent) in those given two doses of toxoid. The superiority of three doses is evident in the 90 per cent reduction of cases, with no deaths, in the 16,829 children given three doses of toxoid in the 1927-1930 period. The estimated reductions in diphtheria agree with the percentages of individuals showing 0.04 unit of antitoxin per cubic centimeter of blood serum, as determined by actual titration, after two and three doses of toxoid.[3]

From the Department of Epidemiology and Biometrics, University of Toronto School of Hygiene.

Read before the Section on Preventive and Industrial Medicine and Public Health at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 13, 1935.

1. Burke, F. S.: The Administrative Control of the Diphtheria Toxoid Campaign in Toronto, 1930, Canad. Pub. Health J., **21**: 209-218 (May) 1930.

2. McKinnon, N. E.; Ross, Mary A., and Defries, R. D.: Reduction in Diphtheria in 36,000 Toronto School Children as a Result of an Immunization Campaign, Canad. Pub. Health J. **22**: 217-223 (May) 1931. Ross, Mary A., and McKinnon, N. E.: The Efficiency of Toxoid in Controlling Diphtheria, Toronto, 1926-1930, ibid. **22**: 333-342 (July) 1931.

3. Moloney, P. J.: Personal communication to the authors.

In 1930 the program of immunization was renewed and is still in progress. Table 2 shows, in summary form, the analysis of the data collected until August 1932 for children given three doses of toxoid. The increment was 14,191 children in 1930-1931 [4] and 13,871 in 1931-1932. The group was therefore of considerable size. When the rates in the controls, schoolmates, corrected for age, for monthly distribution and for difference of susceptibility are applied, as previously, to those given three doses of toxoid, the estimated number of cases for 1930-1931 is 133, the actual cases fourteen, or a reduction of 89 per cent. For 1931-1932 the number of estimated cases is 105, the actual three, a reduction of 97 per cent, and for the two-year period 1930-1932, 238 and seventeen, a reduction of 93 per cent. Over the five-year period of 1927-1932, the estimated number of cases is 460. The actual cases numbered forty, a reduction of 91 per cent. In these forty cases there were no deaths. There is so little variation between these estimates of reduction in diphtheria and between them and the previous estimate that the figure of 90 per cent can be accepted as fairly representative of the efficiency of three doses of toxoid under the conditions of high prevalence of diphtheria obtaining at that time.

But the group each year is not necessarily homogeneous in regard to immunity, as it contains children immunized in the respective year, some the year before, some the year previous to that, and so on. To show the reduction in each year subsequent to immunization, tables 3 to 5 have been prepared.

4. Data are classified by the September through August year.

TABLE 1.—*Reduction in Diphtheria in School Children Subsequent to Toxoid—Toronto Public Schools, 1926-1930*

Doses of Toxoid	Cases: Estimated*	Cases: Actual	Percentage Reduction	Deaths
One dose	34	24	29	3
Two doses	200	52	74	2
Three doses	222	23	90	0

* At rate in controls (schoolmates) corrected for age, monthly distribution, and susceptibility.

TABLE 2.—*Reduction in Diphtheria in Immunized Children—Toronto Public Schools, 1927-1932* *

Year	Cases: Estimated†	Cases: Actual	Percentage Reduction	Deaths
1927-1928	25	1	96	0
1928-1929	84	7	92	0
1929-1930	113	15	87	0
1930-1931	133	14	89	0
1931-1932	105	3	97	0
1927-1932	460	40	91	0

* Given three doses of toxoid.
† At rates in controls corrected for age, monthly distribution, and susceptibility.

TABLE 3.—*Number Immunized and Under Observation—Toronto Public Schools, 1927-1932* *

1927-1928	1928-1929	1929-1930	1930-1931	1931-1932
11,514†	11,039	10,237	9,308	8,099
	4,681†	4,545	4,284	3,907
			14,191†	13,804
				13,871†
(301)‡	(333)‡	(396)‡	(751)‡	
11,514	15,720	14,782	27,783	39,681

* Given three doses of toxoid—46,038.
† The number immunized in each year.
‡ Preschool children immunized previously who arrived at 5 years of age in year indicated.

Table 3 shows the group immunized each year and its subsequent decrease by advancing the age, annually, excluding patients 15 years old from the study and adding a number of children, immunized previously but becoming 5 years of age at the time indicated, and therefore included in the study. It is apparent that the various groups were of a size that might be considered sufficient to give reliable results under usual conditions of diphtheria prevalence and represent a considerable part of the school population of 90,000.

Table 4 shows the diphtheria expectancy, estimated by applying the rates in the unselected controls, corrected for age, for monthly distribution and for difference in susceptibility, and the actual cases in each group each year. It is apparent that in 1931-1932, in spite of a significantly larger group, the expectancy is smaller than previously, so that one is confronted with the uncertainty of small numbers. In spite of this, the percentage reductions in each group have been calculated and are shown in table 5. The estimates for the last two years are more subject to correction than the earlier estimates, on account of the large numbers of children immunized in the later years by the private physicians in the city. These children are included in the control group. Correction for them would give a higher number of estimated cases and therefore a higher degree of efficiency of toxoid. The figures in the last column are, of course, of uncertain significance, owing not to small numbers of children in the various groups but to the paucity of diphtheria in the city, which is disappointing as far as the experimental observations are concerned but very gratifying otherwise. The continued decrease in diphtheria in the nonimmunized as well as the immunized has reduced the expectancy in each group to numbers too small to form a reliable basis for further estimates, by this method, of the efficiency of toxoid. The estimated reduction in table 5 suggests that immunity tends to be highest in the year in which the toxoid is given and that in subsequent years there is a slight decline. But the high level of approximately 85 to 90 per cent efficiency at which immunity is maintained for a period of from

TABLE 4.—*Diphtheria, Estimated and Actual, in Immunized Children—Toronto Public Schools, 1927-1932* *

Given Three Doses of Toxoid in	Diphtheria Cases in 1927-1928: Estimated†	1927-1928: Actual	1928-1929: Estimated†	1928-1929: Actual	1929-1930: Estimated†	1929-1930: Actual	1930-1931: Estimated†	1930-1931: Actual	1931-1932: Estimated†	1931-1932: Actual
1927-1928	25	1	63	7	76	11	67	11	31	0
1928-1929	..	..	21	0	37	4	32	0	14	0
1930-1931	..	..	..	..	..	..	34	3	47	3
1931-1932	..	..	..	..	..	..	..	..	13	0

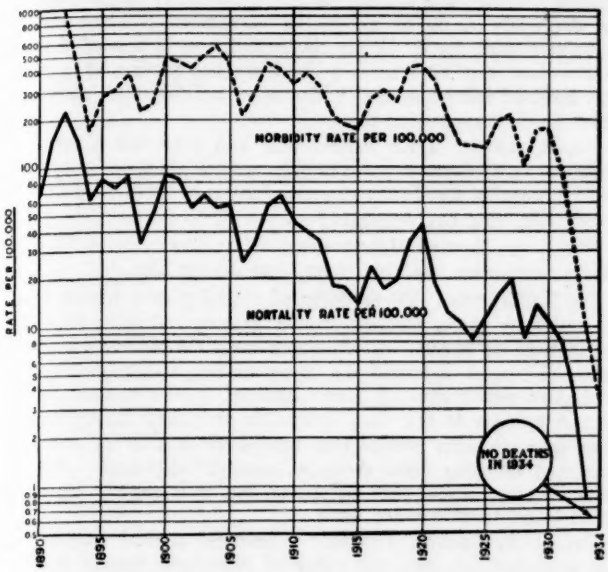
* Given three doses of toxoid.
† Cases estimated by applying rates in controls, corrected for age, monthly distribution, and susceptibility.

TABLE 5.—*Reduction in Diphtheria Subsequent to Immunization—Toronto Public Schools, 1927-1932* *

Given Three Doses in	Percentage Reduction in Diphtheria in 1927-1928	1928-1929	1929-1930	1930-1931	1931-1932
1927-1928	96	89	86	84	100
1928-1929	..	100	89	100	100
1930-1931	..	..	..	91	94
1931-1932	..	..	..	..	100

* Three doses of toxoid.

four to five years, at least, is the outstanding finding in this analysis. It is of real significance that there was not one death in any of the forty cases and, in fact, there has not been to date a death from diphtheria in any of the 46,000 children given three doses of toxoid in Toronto. Evidently the infection has been met in each case with a degree of immunity which, along with



Mortality and morbidity rates per hundred thousand for diphtheria in Toronto from 1890 to 1934.

the usual antitoxin treatment, was sufficient to eliminate any fatalities.

The question of diagnosis of the recorded cases must be considered. Malaise, sore throat and fever were present in all cases. A membrane was present in twenty-eight out of thirty-one from whom information in this regard was obtained; two showed "spots" and one was first diagnosed as Vincent's angina. These three gave "positive swabs." Those from the "spots" were not tested for virulence but that from the case of Vincent's angina was positive.

All but one of the forty cases gave "positive swabs." Of twenty-seven examined for virulence, twenty-six were positive. Even the three cases occurring in 1930-1931 in the group immunized that year were undoubtedly diphtheria. Sore throat, fever and a membrane were present in each. Each gave a positive swab, and two tested for virulence were positive. Two of these cases occurred in the same family within three days of each other, and the third dose of toxoid had been given on the same day four months previously. The diagnoses of diphtheria and therefore occurrence of diphtheria in immunized children must be accepted without question.

It is apparent in the accompanying chart that when the campaign was started diphtheria morbidity and mortality were very high and that for thirty years there had been little if any reduction in morbidity. In 1926 there were more than 1,000 cases with ninety deaths, a mortality rate of 16.2 per hundred thousand. In 1927 the deaths numbered 114, a mortality rate of 20 per hundred thousand. At that time diphtheria was the chief cause of death in the whole age group of 2 to 14 years of age. Under these conditions the campaign was initiated and the first comparisons were made. In 1934 in the same city with a population of 630,000 there were eighteen cases, and for a period of fifteen months, January 1934 to March 1935, there was not one death from diphtheria. This decline has occurred following fairly extensive immunization, and considering the all too small decline in morbidity in the thirty years previously and the established value of toxoid, it is not unreasonable to attribute, for the present at least, the larger part of the decline, directly or indirectly, to immunization.

The data support a not unreasonable hypothesis that a lower degree of immunity, insufficient to control diphtheria completely when it is highly prevalent, is sufficient when the prevalence of diphtheria is low. This is supported, too, by the observations in a group of Schick-negative children and in those with a higher degree of sensitivity to the specific protein, as shown by the reaction test, neither of which were given toxoid, and, too, in children given but two doses of toxoid as shown in table 6. However that may be, modern public health practice requires not only the protection of the community against epidemic disease but also the protection of the indivdual; and for the individual the best possible immunity should be maintained.

Two other pertinent observations may be mentioned: First, Dr. Ambrose Moffat, director of laboratories of the Department of Public Health of Toronto, reports that in 1926 there were 13,231 city swabs examined. Of these, 2,564, or 19.4 per cent, were positive. In 1929 the number examined was 16,542, with 2,754 positive, or 16.6 per cent. In 1934, 4,516 were examined and but twenty-eight, or 0.6 per cent, were positive. In the years mentioned, the cases of diphtheria numbered 1,098, 1,030, and eighteen respectively. Comparison with the number of swabs reveals a much more diligent or widespread collection of swabs in relation to cases in 1934 than in the former years, but in spite of this the number of positives found was practically

TABLE 6.—*Diphtheria in Certain Small Groups—Toronto Public Schools, 1926-1932*

	1926-1927	1927-1928	1928-1929	1929-1930	1930-1931	1931-1932
Schick negatives						
Number under observation	4,855	4,274	4,302	3,596	2,861	2,097
Estimated cases*	10	19	16	17	13	7
Actual cases	1	6	8	2	2	1
3+ reactors to the reaction test†						
Number under observation	1,224	1,008	2,139	1,996	1,477	1,738
Estimated cases*	2	6	9	9	11	6
Actual cases	0	2	2	1	0	0
Children given 2 doses of toxoid						
Number under observation	7,114	6,931	7,316	7,120	6,587	7,113
Estimated cases*	24	58	52	66	63	27
Actual cases	7	10	16	19	3	1
Reduction, per cent	71	83	69	71	95	96

* Estimated by applying rates in unselected controls, corrected for age, monthly distribution, and, for two dose children, "susceptibility." There were two deaths among the "two dose" cases, one death among the "3+ "reactors," and none among the Schick negatives.

† Those showing induration at the site of the test.

The Schick tests were made by competent physicians of the "toxoid teams" in 1926-1927 with toxin which, on distribution, conformed with the requirements of that time; namely, 1/50 minimum lethal dose in 0.1 cc. Such toxin was not entirely free from possible deterioration in handling or from experimental error in diluting. When the cases were classified according to date of testing it was found that, in five instances, there were two cases which had been Schick tested on the same day. This grouping of cases to certain days of testing suggested such deterioration or error. Stabilization of dilute toxin with gelatin has eliminated such inaccuracies so that it is reasonable to expect a higher degree of correlation between the Schick-negative state and actual immunity than is suggested here. It is probable, too, that at least in some instances the individual may have become Schick-positive before the onset of diphtheria. The cases were confirmed as diphtheria by clinical and bacteriologic evidence.

negligible. This is a striking change following thirty years of no significant decline in diphtheria morbidity and suggests that elimination of cases controls the infecting organism or its spread. It helps, too, to lay the bogy of increased danger of carriers.

Second, Dr. G. M. Little of the Red Deer Health Unit in Alberta reports that in 603 children Schick

tested two years after immunization with three doses of toxoid, 98.8 per cent were Schick negative. He adds the very significant information that a Schick-negative reading is a rare finding in that district except following immunization or a history of diphtheria. In that rural community it is very unlikely that latent immunization played any part in the maintenance of that degree of immunity.

SUMMARY AND CONCLUSIONS

In a previous publication covering the period 1927-1930 it was shown that when the corrected rates of diphtheria obtaining in the controls (schoolmates) were applied to 16,829 children given three doses of toxoid in Toronto public schools, the expected cases numbered 222. There were actually 23 cases, a reduction of 90 per cent. There were no deaths in the 23 cases.

Later observations on this group and on 29,029 additional children given three doses of toxoid subsequent to 1929-1930 are reported here. For the whole period 1927-1932, instead of 460 cases, as estimated by applying the rates in controls, there were actually forty cases, a reduction of 91 per cent. In each year the reduction approximated this figure. To date no death from diphtheria has occurred in any of the 46,000 children given three doses of toxoid.

In dividing the data so as to follow the reduction of diphtheria in each year subsequent to immunization, there is some evidence that the reduction is greatest and therefore immunity greatest in the year of immunization, that in subsequent years there is a slight decline, but that immunity remains at a very high level, from 80 to 90 per cent, for at least four or five years.

The decline in diphtheria in the nonimmunized as well as immunized vitiates comparisons of later data.

Diphtheria morbidity rates in Toronto had shown no definite decline over a period of thirty years. In 1926 and 1927 the mortality rates, quite typical of that period, were 16.2 and 20 per hundred thousand. Following the progress of immunization, diphtheria has fallen so that in 1934, in a city of 630,000 population, there were but eighteen cases and for a period of fifteen months there was not one death. In 4,516 swabs examined in 1934 from suspected cases, carriers and contacts, only twenty-eight, or 0.6 per cent, were positive. This change in diphtheria in Toronto suggests that reduction of cases in immunized children reduces the cases in the nonimmunized and that the control of cases controls also the infecting organism or its spread.

POSTSCRIPT.—September 18, since this paper was written, a death occurred from diphtheria in a child 6½ years of age, who was given three doses of toxoid five years previously at 1 year of age.

ABSTRACT OF DISCUSSION

DR. M. P. RAVENEL, Columbia, Mo.: The precipitated toxoid was made first by Glenny in England and was popularized in this country by the late Dr. Leon Havens of Alabama and carried on by Dr. Baker and others. I think I am right in saying that in America we think the use of toxoid is a little behind the times. We have used chiefly the alum precipitate. I have been trying to find out why the Connaught Laboratories do not approve of the alum precipitate, and why Canada continues to use the toxoid. I have asked Dr. McCoy of the National Institute of Health and other authorities, and nobody knows why Canada has adhered to the toxoid.

DR. N. E. MCKINNON, Downsview, Ont.: I tried to indicate that the reduction in diphtheria was comparable with the number of individuals showing one twenty-fifth unit of antitoxin per cubic centimeter of blood serum. Three doses of toxoid will give from 90 to 100 per cent immunity in those who get one twenty-fifth unit of antitoxin. With the number of cases occurring in children, immunity is necessary. Nothing else is entirely satisfactory. I don't know of any other product or procedure that will give that degree of immunity. Of course we do get cases in children giving Schick negative reactions, and they are diphtheria. They have membrane, fever, malaise, sore throat, and, when we get swabs from them, they are causative and, when swabs are tested for virulence, they are positive; so they are diphtheria, and there is no question about the diagnosis of diphtheria in the immunized children giving negative Schick tests. While the Schick test is a good test, it isn't a final test of immunity, and we are aiming at getting that level of immunity and will not be satisfied with anything less. Does that answer the question?

DR. RAVENEL: I am asking why you don't use alum precipitate. It produces as good effects as multiple doses of toxoid, and I think that Dr. Baker can give figures of some 18,000 cases which will bear out my assertion.

DR. J. N. BAKER, Montgomery, Ala.: Between 16,000 and 18,000 cases were reported more than a year ago.

DR. MCKINNON: Three doses of toxoid give a higher level of antitoxin and a correspondingly greater reduction in diphtheria than do two doses. As some cases do occur after three doses of toxoid, it would not seem advisable to advocate any procedure which gives a lower level of antitoxin, and, therefore, according to our data, a smaller reduction in diphtheria. One dose of alum precipitated toxoid gives a lower level of antitoxin than do three doses of unmodified toxoid. Schick tests may not reveal this difference, but it is readily apparent when blood titrations are done.

DR. D. T. FRASER, Toronto: The most critical comparison for assessing the antigenic value of diphtheria toxoid in man is to use one given lot or batch of toxoid, of which one portion was prepared as alum precipitate, the other portion unmodified. The number of flocculating units per cubic centimeter in each portion was adjusted so that they were equal; namely, twenty. These preparations were used on a homogeneous group from whom blood had been drawn and whose serum was shown initially to possess no detectable antitoxin (less than one five-hundredth unit). Subsequent titrations of antitoxin were made at intervals, after injection of toxoid, to measure the response of each individual. The question as to whether one dose of alum precipitated toxoid will effectively protect against diphtheria can be determined only by an experience covering five or six years and then subjecting the data on immunization and subsequent diphtheria to a critical statistical analysis. Meanwhile, we have been interested in comparing only the response in antitoxin to one dose of alum precipitated toxoid with three doses of unmodified toxoid. Our groups at the present time are admittedly small: forty and thirty-five. In the one dose (alum toxoid) group, after ten weeks, twenty-five, or 62 per cent, had more than one one-hundredth unit; in the three dose (unmodified toxoid) group, ten weeks after the first dose, thirty-two, or 91 per cent, had more than one one-hundredth unit. After one year, in the former group, 19 per cent had more than one one-hundredth unit; in the latter group, 91 per cent. The individual titers in the one dose alum group were very much lower than were those in the three dose unmodified toxoid group. After one year, though individually the titers had dropped in both groups, none had dropped below one one-hundredth unit in the three dose group, whereas fifteen of the thirty-six tested had dropped below this figure in the one dose group. The choice of one one-hundredth unit per cubic centimeter of serum is somewhat arbitrary, but it is approximating the Schick level. Persons possessing one one-hundredth unit are Schick negative; those with less than one two hundred and fiftieth unit are Schick positive. We interpret our observations as indicating that one dose of alum is inferior to three doses of unmodified toxoid.

DR. BAKER: In 1931 and 1932 the director of the Alabama laboratory, Dr. L. C. Havens, carried out experimental work in trying to determine how the three-dose toxoid might be reduced to one dose and yet give the desired immunity, thereby simplifying the technic of immunization as well as the labor, in mass application of the immunizing agent. We worked on this clinically and steadily for over a year. The one-dose alum precipitated product was used in one county in

something like 1,000 cases. This study revealed about 95 per cent, at the end of a period of from three to six months, to be Schick negative. Shortly after this work in Alabama, Dr. McGinnes of Virginia became interested in the possibilities inherent in the one-dose product and conducted many series of studies, which were carefully checked, with the one-dose toxoid, and with results even more conclusive than those procured in Alabama. At the 1934 meeting of the American Public Health Association the work done both in Virginia and in Alabama was submitted somewhat in detail. Also at the 1933 meeting of the American Public Health Association I submitted an outline of the work which had been done up to that time in Alabama, with the one-dose toxoid. Within the past two years many of the biological houses have been marketing this one-dose product. Apparently there has not been uniformity of procedure in the preparation of these products, and as a consequence occasional untoward effects have resulted. It is felt by our laboratory workers in Alabama that a rigid adherence to the technic recommended by them would obviate such complications in a large measure and we have so advised. My feeling is that the one-dose alum precipitated toxoid rests today on solid scientific and clinical ground and that it, or some modification of it, is here to stay.

DR. N. E. MCKINNON: One dose of alum precipitated toxoid is much better than one dose of unmodified toxoid, and, if under certain circumstances only one dose can be given, the alum toxoid should be used. Those using it should be aware, however, that the resulting immunity is definitely less than that produced by three doses of unmodified toxoid.

DEMENTIA PARALYTICA AND TABES

A STUDY WITH REFERENCE TO PRECOCIOUS DEVELOPMENT

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The age distribution of patients with dementia paralytica and tabes dorsalis as observed in this clinic has afforded the basis for this study. A large number of cases have occurred under the age of 30 years in the group of dementia paralytica patients of all ages. This observation was commented on by Felix Plaut,[1] Josef Jadassohn,[1] L. M. Pautrier[1] and Bruno Bloch[1] during their visits to this clinic. The age incidence was apparently much higher in their clinics in Europe, and the occurrence of dementia paralytica in acquired cases of syphilis in patients under 30 years of age was regarded by them as unusual.

It was thought important to compare this group of cases with a series of tabetic cases seen over exactly the same period and under the same conditions.

Accordingly, there are available for study over a period of eight years, from July 1925 to July 1933, 436 cases of dementia paralytica and 378 cases of tabes. Of this number, seventy-seven cases, or 18.3 per cent, occurred in dementia paralytica patients under 30 years of age, and twenty-one tabetic patients, or 5.5 per cent, fall in the group below 30 years of age. All the cases presented in this study were frank clinical examples of the two syndromes in which the diagnosis was substantiated by typical changes in the spinal fluid. Cases of juvenile dementia paralytica and tabes were excluded from this study.

The entire group of patients presented herein represents 48.1 per cent of all patients in whom a diagnosis of neurosyphilis was made, 25.6 per cent of whom had dementia paralytica and 22.5 per cent tabes. This discrepancy is a fair indication of the ratio of frequency of these two divisions of neurosyphilis.

Dementia paralytica from acquired syphilis occurring in a young age group has been reported by Kirschbaum.[2] This observer records thirty-six cases of dementia paralytica in individuals under 30 years of age, representing from 3 to 4 per cent of his entire dementia paralytica material. He also found an average elapsed period of five years from the date of infection to the onset of symptoms in the young group. This figure corresponds to our own of 5.34 years for the same group. However, our age incidence in the younger group is six times as high as that recorded by Kirschbaum.

In seeking causative factors for the early occurrence of cortical degeneration in so young a group, Kirschbaum suggests poor protoplasm (constitution) and excessive indulgence in alcohol as contributing factors. In connection with the latter factor, it is interesting that our study entailing a much higher percentage of cases occurred during the period of national prohibition. While alcohol was clandestinely procurable during this period, we failed to find it a factor of major importance. Kirschbaum further states that therapy—good, indifferent or bad—was not a factor in the shortness of the period of latency. Another finding of interest in his study which is definitely borne out in our own is a greater degree of mental deterioration in the younger group than occurred with older dementia paralytica patients.

The age incidence of dementia paralytica at St. Peter State Hospital, St. Peter, Minn., was reported by Petersen.[3] He reports an age variation of from 20 to 77 years in his cases and found a predominance of males in all decades except in the decade from 20 to 30. He reports 6 per cent of 179 cases of dementia paralytica as occurring under 30 years of age, which is one third as many as has been found in our series.

For purposes of comparison, the cases of dementia paralytica and tabes are divided into those under and those over 30 years of age. These two groups will hereafter be referred to as the younger and the older groups. These groups are compared with regard to the age, sex, occupation, nationality, duration of infection and previous treatment given the patient before the onset of symptoms. The mental changes encountered in the series of dementia paralytica patients and the symptomatology of the tabetic patients are commented on in both age groups.

AGE AND SEX

The age and sex distribution of the entire group of cases is shown in table 1. It is seen from this table that the majority of cases of dementia paralytica in both sexes occurred between the ages of 30 and 50 years, while the majority of tabetic cases occurred between 40 and 60. In dementia paralytica 18.3 per cent of the cases occurred under 30, compared with only 5.5 per cent of cases of tabes occurring under 30, whereas 16.3 per cent of cases of dementia paralytica

Studies and Contributions of the Department of Dermatology and Syphilology, University of Michigan Medical School, service of Dr. Udo J. Wile.
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1. Personal communication to the authors.

2. Kirschbaum, W.: Ztschr. f. d. ges. Neurol. u. Psychiat. **137:** 552-559 (Nov.) 1931.

3. Petersen, W. F.: Am. J. Syph. & Neurol. **18:** 75-91 (Jan.) 1934.

and 38.5 per cent of tabes occurred over the age of 50. It is at once apparent that the precocity of development is greater in dementia paralytica and the age onset of tabes is considerably higher than that of dementia paralytica. It is interesting to note that there were twice as many tabetic as compared to dementia paralytica patients over 50 years of age and that there were three times as many young dementia paralytica patients as

TABLE 1.—*Age and Sex Distribution in 436 Cases of Dementia Paralytica and 378 Cases of Tabes Dorsalis*

	Males		Females		Totals	
Age	Dementia Paralytica	Tabes	Dementia Paralytica	Tabes	Dementia Paralytica	Tabes
16–20	0	0	4	0	4	0
20–30	51	16	22	5	73	21
30–40	103	79	20	16	123	95
40–50	135	100	28	18	163	118
50–60	43	90	16	15	59	105
60–70	12	28	2	9	14	37
70–80	0	2	0	1	0	3
Totals	344	314	92	64	436	378
						814

there were young tabetic patients. With the average duration of the infection found to be practically the same (15.6 years for dementia paralytica and 16.8 years for tabes), this finding appears to be significant. It perhaps indicates a greater longevity for patients with tabes.

In the analysis of sex, a predominance of males is found in all groups. The ratio for the entire group of dementia paralytica is 3.7 : 1, while in tabes it is higher, being 5 : 1. This ratio is distinctly altered in the younger group, in which a ratio of 2 : 1 in dementia paralytica and 3 : 1 in tabetic patients was found. The ratio of the older group does not differ so markedly, that of dementia paralytica being 4.4 : 1 and tabes being 5 : 1.

The age and sex distribution of the younger groups is shown in table 2.

The average age of the young dementia paralytica patient was 26.1 years; of the young tabetic, 27 years. It is noteworthy that, while four patients with dementia paralytica were under the age of 20 and seventeen were from 20 to 25 years of age, no tabetic patient was seen below the age of 22 years and only five between the ages of 22 and 25.

Of outstanding importance in the analysis of sex is that all four patients with dementia paralytica under 20 were women and that there were six women in the seventeen patients between 20 and 25 years of age. In tabes, the youngest patient seen was a woman, aged 22. Only four other patients were seen below the age of 26, all of them being men. The occurrence of a higher percentage of females in the younger group of dementia paralytica as compared to the older group concurs with the observations of Petersen.[3] From these statistics it can be seen that dementia paralytica affects the female sex in greater proportion than does tabes, thus bearing out the observations in "Cooperative Clinical Studies in the Treatment of Syphilis";[4] i. e., the female bears a greater immunity to tabes than to dementia paralytica. However, it is significant that in both dementia paralytica and tabes a proportionately greater number of females appeared in the younger group than in the older. Sex, therefore, appears to constitute a predisposing factor in our younger group.

4. Clark, Taliaferro; Parran, Thomas, Jr.; Cole, H. N.; Moore, J. E.; O'Leary, P. A.; Stokes, J. H., and Wile, U. J.: Cooperative Clinical Studies in the Treatment of Syphilis, Ven. Dis. Inform. **13**: 135, (April 20) 1932. Stokes, J. H.; Cole, H. N.; Moore, J. E.; O'Leary, P. A.; Wile, U. J.; Clark, Taliaferro; Parran, Thomas, Jr., and Usilton, Lida J., ibid. **13**: 165 (May 20), 207 (June 20), 253 (July 20) 1932.

OCCUPATION AND NATIONALITY

In searching to find further predisposing causes, the occupation, intellectual background, nationality and race of the individual were studied in the younger groups and compared to the same factors in the older groups.

An analysis of the occupations of all dementia paralytica and tabetic patients revealed no essential differences. No data were available to account for either the youthfulness of the patient or the precocity of development of the neurosyphilis. For this reason, this factor will be studied collectively in the two age groups of both dementia paralytica and tabetic patients.

It has been the consensus of most syphilographers and psychiatrists that dementia paralytica affects a considerably greater percentage of the educated strata of society than does tabes. Of particular interest in our series of cases was the predominance of the nonprofessional class in both dementia paralytica and tabes. This class constituted 65.58 per cent of all dementia paralytica and 70.4 per cent of all the tabetic patients. Only 4.5 per cent of all dementia paralytica patients and 3.5 per cent of all tabetic patients were of the professional class. The remaining percentages in both dementia paralytica and tabetic patients were about equally divided between housewives and individuals with no given occupation.

From this study it seems that occupation and intellectual background apparently play no important rôle in the occurrence of dementia paralytica or tabes. McKinlay,[5] in a survey of dementia paralytica and tabes, found that the laboring class is the greatest affected in both diseases and that the intelligence quotient does not necessarily predispose to either.

Comparative study of the nationalities of the two age groups of dementia paralytica reveals a distinct difference. In the older groups, 20 per cent of dementia

TABLE 2.—*Age and Sex Distribution in Younger Groups of Dementia Paralytica and Tabetic Patients*

	Males		Females	
Age	Dementia Paralytica	Tabes	Dementia Paralytica	Tabes
16	0	0	1	0
17	0	0	1	0
18	0	0	0	0
19	0	0	2	0
20	1	0	1	0
21	1	0	0	0
22	3	0	0	1
23	2	0	1	0
24	1	1	2	0
25	3	3	2	0
26	7	4	4	1
27	10	0	4	1
28	9	1	4	1
29	14	7	4	1
Totals	51	16	26	5

paralytica and 29.7 per cent of tabetic patients were foreign born. By contrast, in the younger groups, 3.8 per cent of dementia paralytica and 28.5 per cent of tabetic patients were not natives of this country. Summed up, therefore, a larger percentage of tabetic patients than of dementia paralytica patients were foreigners, foreign predominance being even more marked in the younger group.

Data as to the source of the infection, whether foreign or native, were not available. The foreign-born dementia paralytica patients represent twenty-one dif-

5. McKinlay, P. L.: J. Hyg. **28**: 394-417 (Feb.) 1929.

ferent nationalities, and twenty-four nationalities are represented in the tabetic group. A possible significant feature, however, is that one third of the foreign group of dementia paralytica patients were native Greeks and Italians, while only one seventh of the similar group of tabetic patients were of these races.

It appears, therefore, from the data presented from the comparative study of the two age groups that dementia paralytica developed earlier in native-born patients than in those of foreign extraction, while this was not found to hold true for tabes. Conversely, it appears from this study that foreigners are more susceptible to cord involvement than to cortical involvement.

DURATION OF THE INFECTION

The presence of mental deterioration in the first examination of patients with dementia paralytica leaves the history of infection for the most part in obscurity. In a number of other cases in which a history could have been given, the infection, as so frequently happens, was an occult one and knowledge of the infection was denied. However, since mental deterioration is not part of the tabetic syndrome, a larger number of tabetic patients with accurate histories of infection might be expected. The accuracy of history is brought out in the following figures: Forty-four per cent of dementia paralytica in contrast to 55 per cent of tabetic patients of the older group knew of early manifestations. An accurate history of infection was determined in 33 per cent of the younger dementia paralytica patients and in 42 per cent of the corresponding group of tabetic patients.

The elapsed time between the date of the infection and the onset of symptoms in the older group of dementia paralytica patients varied from two to forty-two years and averaged 15.6 years. The corresponding latent period in a similar group of tabetic patients varied from one to forty-nine years and averaged 16.8 years. An onset of symptoms within five years after infection was found in thirteen dementia paralytica and fourteen tabetic patients of the older groups. It is apparent from these figures that we are dealing with a certain group of older patients who show a precocious onset of symptoms. This finding, however, does not bear on the development of dementia paralytica or tabes so far as the youthfulness of the patient is concerned.

The elapsed time between the date of the infection and the onset of symptoms in the younger group with dementia paralytica varied from six months to thirteen years and averaged 5.34 years, while in the young tabetic group it varied from two to eleven years and averaged 6.01 years. The elapsed time in the younger group with dementia paralytica is one third as long as in the older group, while it was only slightly less than one third in tabes.

One of us (U. J. W.) in his personal experience has seen dementia paralytica develop within the first year after infection and result in death established at autopsy within two years. Kirschbaum [2] found no cases with an elapsed time of less than three years in his series of cases. The patient in our study with an elapsed time of six months had clinical dementia paralytica. He was given six intravenous injections of arsphenamine (elsewhere) within the first six weeks of the infection. When he presented himself at this clinic six months after the infection, spinal fluid changes substantiating the clinical diagnosis of dementia paralytica were found. In addition, he was slightly deteriorated mentally. This patient was kept under observation for ten months while receiving treatment. The spinal fluid changes remained abnormal and there was no improvement in the mental status of the patient.

Because of the youthfulness of the patient and the precocity of onset, the following case report is given. Difficulty in walking and a sensation of numbness brought the youngest tabetic patient to the clinic. The duration of his infection was two years. Examination revealed round, equal and reactive pupils, loss of lower reflexes and a typical ataxic gait. The serologic reaction of the blood was positive, and examination of the spinal fluid substantiated the clinical diagnosis. During the first two years following infection the patient received 100 mercury rubs and about 500 Gm. of potassium iodide elsewhere. While under treatment at this clinic he received twelve intravenous arsphenamine, four tryparsamide and forty-eight bismuth salicylate injections and twelve malarial paroxysms. When last seen the patient was subjectively improved, having only occasional pains in the legs. Examination, however, after this lapse of time and treatment showed Argyll Robertson pupils, absent lower reflexes, swaying in the Romberg position, early optic atrophy, and an essentially normal spinal fluid.

In reviewing the entire group of both older and younger patients it was found that one dementia paralytica patient out of every five as contrasted with one tabetic patient out of every eleven showed a precocious onset of symptoms. With regard to the age distribution, dementia paralytica developed in one patient out of 5.6 of the entire group before the third decade, while only one tabetic case out of eighteen of the entire group occurred under 30 years of age. Comparison of these data reveals a striking difference, showing the precocity of development of dementia paralytica to be much greater than that of tabes.

PREVIOUS TREATMENT

With regard to previous treatment as bearing on the protection of the individual against the development of dementia paralytica and tabes, there are available for study 160 cases in the older age group of dementia paralytica and 196 cases in the corresponding tabetic group. As previously stated, the date of the infection was noted in all these cases. The type of treatment and the time at which the treatment was administered in relation to the age of the syphilis have been analyzed.

It was brought out by Moore and his associates [4] that the development of neurosyphilis was more frequent in those patients receiving poor treatment than in those who received no treatment. As bearing remotely on this, our patients, both dementia paralytica and tabetic, who received poor therapy following infection developed neurosyphilis sooner than those who had received no previous treatment.

In "Cooperative Clinical Studies in the Treatment of Syphilis," it was found [4] that the progression of neurosyphilis was much greater in patients receiving poor treatment than in those who received relatively good therapy. The protection afforded by adequate previous treatment, however, is demonstrated by the fact that only two dementia paralytica patients of the entire 160 and only four tabetic patients of 196 received adequate previous treatment. The remaining patients received poor, irregular or no treatment. The last figure bears out the view that inadequate or no treatment is a predisposing factor in the development of dementia paralytica and tabes.

In the younger group there are only twenty-six dementia paralytica and ten tabetic patients giving his-

tories of infection. These cases are so few as to render statistics unreliable. However, the percentage of cases in both younger groups receiving inadequate or no treatment was essentially the same as in the older group; thus bearing out the previous observations.

COMMENT

The incidence of marked mental deterioration in the younger group of dementia paralytica patients was higher than in the older group and, furthermore, the degree of deterioration was much more marked. Thirty-five, or almost one half of the patients in the younger group, showed varying degrees of deterioration at the first examination. This compares with but 32.2 per cent of the older group that were mentally deteriorated. These observations would not tend to corroborate those of Caldwell,[6] who states that the prognosis in the younger dementia paralytica patients is better than in the older ones.

The mental changes noted particularly in the younger group were the early onset of dysarthria, disorientation, euphoria and at times irritability. Factors that affect the neuropsychiatric condition of the individual, such as accidents and economic losses, appeared to precipitate the onset of symptoms.

There was no essential difference noted in the severity of tabes between the younger and older groups; i. e., age does not seem to be a factor in the severity of the disease.

CONCLUSIONS

1. In 436 cases of dementia paralytica and 378 cases of tabes studied with special reference to precocious development, the onset of symptoms occurred earlier in dementia paralytica than in tabes.
2. Dementia paralytica occurred more frequently in females than did tabes.
3. Of four patients in whom the dementia paralytica syndrome occurred before the twentieth year, all were women.
4. Occupation and intellectual background played no important rôle in the occurrence of dementia paralytica and tabes in this series.
5. Tabes occurred more frequently than dementia paralytica in the foreign born in this study.
6. Precocity of onset was found more frequently in dementia paralytica than in tabes.
7. As has been shown for neurosyphilis in general, the absence or inadequacy of treatment was a predominant factor in the precocious development of both dementia paralytica and tabes.
8. In the young group, severity of symptoms seemed more marked in dementia paralytica than in tabes.

ABSTRACT OF DISCUSSION

DR. A. BENSON CANNON, New York: I am impressed with the great number of cases of dementia paralytica and tabes that have been treated in this comparatively small community. In the hospitals with which I am associated in New York we find that there is a definite decrease in the number of cases with neurosyphilis, especially with cases of dementia paralytica and tabes. Only a few days ago, one of the attendants at the New York Neurological Institute spoke of the difficulty of getting a sufficient number of cases of tabes and dementia paralytica to demonstrate to the students. In recent years, neurologists have found it necessary to specialize in vascular diseases of the nervous system because of the scarcity of cases of neurosyphilis. The age distribution of dementia paralytica and tabes in the cases of Drs. Wile, Poth and Barney is unusual. In studying the number of cases admitted to the Vanderbilt Clinic and City Hospital for the past three years and combining these figures with those from the Neurological Institute for the past five years, we find 528 cases of dementia paralytica and tabes combined. Of this number we have about 299 cases of tabes, sixty-six of which were in women. There were 229 cases of dementia paralytica and only forty-one of these were in women. There was only one patient less than 21 years of age and that was a girl having dementia paralytica. In going over the records of our cases of dementia paralytica, we had to change many of the diagnoses because of the patients' recovery. We have records of many cases showing typical dementia paralytica symptoms both clinically and serologically, diagnosed as dementia paralytica by competent neurologists. Some of these patients had even been confined in institutions, yet under adequate treatment they became clinically and serologically cured and have remained so for from seven to eight years. I feel that it is impossible sometimes to distinguish between dementia paralytica and meningovascular syphilis, and the results of the tests of the spinal fluid do not aid us in making the differentiation. A dementia paralytica curve means only that and does not necessarily signify that the patient has dementia paralytica. I also think that acute cerebrospinal meningitis of the basilar type is often hard to distinguish from dementia paralytica. Our own observations are in agreement with the author's; that is, our patients with tabes and dementia paralytica either had no previous treatment or else inadequate medication and I thoroughly believe with him that, if all patients with early syphilis had the proper standard treatment in the beginning, there would be few or no patients with neurosyphilis.

DR. UDO J. WILE, Ann Arbor, Mich.: The figures brought out in this study are a little difficult to understand if one has a rigid picture of the natural history of syphilis. However, if one takes a broad biologic view of syphilis as a disease constantly changing like other chronic infections these figures are not so difficult to understand. Most of us are educated in medicine with the concept that neurosyphilitic cases of the parenchymatous types are always late sequels of the disease. This has come down to us more or less as a tradition based on the facts and the character of syphilis as it existed two or three decades ago. Those of us who are able to make careful hospital study, together with the autopsies of syphilitic material, have seen this disease gradually modifying its picture, and it is safe to predict that the next two or three decades will see the disease changing in its manifestations from the present-day picture. The material in this study establishes the fact that severe parenchymatous disease of the brain occurs not infrequently early in syphilis, contrary to the generally accepted belief that it is always a late sequel. The differences established in point of time between the onset of tabes and dementia paralytica are not difficult to understand. While we generally place these two conditions more or less together, it is nevertheless a fact that they are distinctly different processes. In dementia paralytica we are dealing with a frank syphilitic encephalitis with many organisms and with active syphilomas and late degeneration, while in tabes the process is a simple degenerative one, and in well established cases little residual syphiloma is present and few, if any, organisms. The rapid onset of dementia paralytica and tabes as established in this paper leads to interesting speculation. It cannot be due, at least in this group, to the influence of arsphenamine or in any way connected with arsphenamine therapy, as the majority of patients here concerned had little or no treatment. If precocity of development is a new phenomenon in neurosyphilitic incidence, it may be explainable on the basis of a change in the natural history of the disease, always a possible factor in any morbid process; or it may be due to the stress and strain of life incident to the present period. These two factors may, of course, be concerned. At any rate we feel certain that under existing conditions severe neurosyphilitic accidents exemplified in dementia paralytica and tabes occur much earlier in the course of the disease than is generally known and accepted as fact.

DR. S. B. HADDEN, Philadelphia: We are engaged on a similar review of cases in the special clinic for the treatment of neurosyphilis at the Philadelphia Hospital—a series of slightly more than 1,500—and I can appreciate the hours that

6. Caldwell, W. A.: Brit. M. J. **2**: 1129 (Dec. 19) 1931.

the authors have spent on this problem. Although our conclusions are far from being drawn, we have proceeded far enough in the work to have formed several opinions, which are in accordance with those of Dr. Wile and his co-workers. It has been an observation in the department for a long time that the age of onset in dementia paralytica is much earlier than in tabes, and I agree with the reasons given for this by Dr. Wile. In reviewing this group we have noted another factor that enters into the late age incidence of the tabetic patient. The tabetic patients represent a relatively untreated group. The dementia paralytica patients have received considerably more treatment. In the two groups, the onset has been premature in the patients who have received inadequate treatment before the development of secondary eruptions. We feel that the syphiloderm stimulates a mechanism that protects the central nervous system. A review of the admissions of dementia paralytica patients in two five-year groups 1914 to 1919 and 1924 to 1929 has shown an increase of 549 per cent. These periods are used because routine spinal fluid examinations were made in practically all cases. We have noted in recent years a remarkable reduction in the incidence of aneurysm not only in the neuropsychiatric division but in the medical division as well. While aneurysm has been definitely decreasing in its frequency since the introduction of the arsphenamines, our admissions of dementia paralytica and tabes patients are steadily increasing. Inadequate treatment may be the answer.

DR. DUNCAN O. POTH, Ann Arbor, Mich.: The group of cases presented in the study occurred over an eight-year period. When these cases were called for from the record room, we were presented with more than 1,200 records of cases in which the diagnosis of dementia paralytica had been made. Out of this group we accepted 436 in which we feel that the diagnosis of dementia paralytica was clear and concise.

THE DEVELOPMENT OF THE THERAPEUTIC USE OF FORCED PERIVASCULAR (SPINAL) DRAINAGE

GEORGE M. RETAN, M.D.
SYRACUSE, N. Y.

In 1931 I published an article on the treatment of infections of the central nervous system by forced spinal drainage, describing cases of poliomyelitis, acute encephalitis, septic meningitis and syphilitic meningitis. These cases were treated by spinal drainage coincident with the intravenous injection of hypotonic solution. These were the first cases treated by this method to be described in the medical literature.

The term "forced spinal drainage" has led to some misconception as to the essential factor involved in this treatment. The term suggests that drainage of the spinal fluid is essential. If this were true, the amount of spinal fluid produced in a certain case would have some relation to the therapeutic result. This has not occurred. I have therefore suggested the term, "forced perivascular drainage," to emphasize better one of the chief factors.

THE PHYSIOLOGIC BASIS OF THE TREATMENT

Starling [1] in 1909 published his studies on the formation of lymph. The essential facts as related to this subject are as follows:

Osmotic equilibrium between the blood stream and the pericellular fluid spaces of the body is maintained by the action of the nondiffusible colloids of the blood and the crystalloids. Outside of the capillary wall the crystalloids are more concentrated than are the crystalloids of the blood stream, tending to balance the action of the nondiffusible colloids. Fluid movement through the capillary wall is therefore mainly produced by the increased blood pressure exerted in the arterial capillary. Passing through the arterial capillary to the venous side, the nondiffusible colloids are able to pull fluid into the venous capillary.

Water of metabolism, the breaking up of particles into smaller molecules, thereby multiplying their osmotic force, and certain electrical charges also influence this force.

Weed and McKibben [2] in 1919 found that the injection of hypotonic solutions into the blood stream of animals produced hydration of the perivascular fluid spaces in the brain and cord and widening of the perivascular fluid pathways.

In 1928 Kubie [3] further demonstrated that, if a needle is introduced into the subarachnoid space and the cerebrospinal fluid allowed to drain, no hydration of the brain or spinal cord results either in gross or in microscopic sections.

DEVELOPMENT OF THE APPLICATION OF THESE PRINCIPLES

Spurling [4] in 1928 published a report on eight cases of infections of the central nervous system in which he performed a lumbar laminectomy and gave quantities of water by mouth. He described the treatment in three cases of septic meningitis and one case of poliomyelitis with bulbar involvement.

In 1930 Freemont-Smith [5] reported a group of cases of multiple sclerosis treated by continuous drainage coincident with the ingestion of quantities of water by mouth. As a part of the treatment he injected pitressin at intervals.

In 1930 Casten [6] reported a case of tryparsamide amblyopia, treated by a two hour drainage of cerebrospinal fluid and 2 liters of water by mouth. He gave 50 cc. of doubly distilled sterile water intravenously and one ampule of solution of pituitary. The patient was improved.

The results of clinical research of this character are difficult of interpretation. The history of human immune serum in the treatment of preparalytic poliomyelitis illustrates this. The two diseases best adapted to a study of this kind are Sydenham's chorea and poliomyelitis in the monkey. In chorea marked and spontaneous improvement rarely occurs from one day to another, and modification of this disease by treatment is significant. In experimentally produced poliomyelitis, by employing intracerebral inoculations of virulent virus, a disease can be produced that will cause complete skeletal paralysis and death in all inoculated animals. Modification of the disease thus produced might lead to more accurate interpretation. Results can be compared with untreated controls.

In developing this treatment my aim has been to study the therapeutic effect of certain phases and factors involved, in the hope of determining the type of procedure that will produce the best therapeutic results.

Read before the Section on Pediatrics at the Eighty-Sixth Annual Session of the American Medical Association, Atlantic City, N. J., June 14, 1935.

1. Starling, E. H.: The Fluids of the Body, London, Constable & Co., Ltd., 1909.

2. Weed, L. H., and McKibben, P. S.: Am. J. Physiol. **48**: 512, 531 (May) 1919.

3. Kubie, L. S.: Brain **51**: 244 (June) 1928.

4. Spurling, R. G.: Kentucky M. J. **26**: 242 (May) 1928.

5. Freemont-Smith, Frank; Putnam, T. J., and Cobb, Stanley: Forced Drainage of the Central Nervous System, Arch. Neurol. & Psychiat. **23**: 219 (Feb.) 1930.

6. Casten, Virgil: New England J. Med. **202**: 676 (April 3) 1930.

Certain factors are controllable, others uncontrollable. The controllable factors are:

1. Concentration of salt in the intravenous solution.
2. Rate of injection per pound hour.
3. Length of time of the injection.
4. Interval between treatments.
5. Blood volume.
6. Possible use of the hydration factor.
7. Use of pitressin or solution of pituitary.

The uncontrollable factors are:

1. The existence of inflammatory tissue within the central nervous system.
2. The existence of inflammatory tissue outside the central nervous system.
3. Kidney function.
4. Cardiac reserve strength.
5. Vascular tone.

CONCENTRATION OF SALT SOLUTION

The degree of change in the osmotic pressure produced by the intravenous injection would depend on the concentration of the salt solution and the rate of injection. The maximum effect would be obtained by a salt solution just above the point at which hemolysis of red cells would be produced. All my earlier work was done with 0.45 per cent sodium chloride, and I did not lower the salt percentage because I was obtaining good clinical results. A case of chorea that did not improve after two treatments with 0.45 per cent sodium chloride showed definite improvement when 0.375 per cent sodium chloride was given. Since that time I have mostly used this solution. I have been in the habit of testing the fragility of the red cells. This is not necessary, as the osmotic pressure of the blood stream is not lowered to the point represented by 0.375 per cent sodium chloride. I have failed to find any evidence of hemolysis in the cases tested. In a few cases in human beings I have used a 0.35 per cent solution but have never gone below this concentration.

Monkeys with poliomyelitis have not recovered when treated with 0.45 per cent sodium chloride, although several have recovered when 0.375 per cent sodium chloride was used. In one monkey in which we used a 0.35 per cent solution my associates and I found evidence of hemolysis of the red cells. However, this solution was given at too rapid a rate. At the present time we are doing all our poliomyelitis research in monkeys with 0.375 per cent sodium chloride. I believe that more experimental work needs to be done before it is known just what solution is best.

RATE OF INJECTION PER POUND HOUR

It would be desirable to determine the amount of hypotonic solution per pound of body weight to be injected each hour. The approach to this problem lies, first, in the effect of the rate of injection on blood volume, and, of greater importance, in the study of the therapeutic result. The latter offers difficulties. The movement of fluid within the body during the injection of massive doses of hypotonic solution varies a great deal in individuals and also in the same individual at different times. Monkeys treated with intravenous rates of less than 9 cc. per pound hour have not recovered. However, monkeys in which injections averaging from 9 to 11 cc. per pound hour, with 0.375 per cent sodium chloride have been used have recovered without paralysis. The blood volume has not been greatly increased during an eight hour injection of 0.375 per cent sodium chloride given at this average rate. This, however, cannot be exactly translated to human beings.

For example, I do not believe that one could safely give a 100 pound (45.4 Kg.) person 1 liter an hour for an eight hour period. The rate of injection per hour also depends on the number of hours that the injection is to be given; I have given a baby with acute encephalitis a four hour treatment at an average rate of 19 cc. per pound. The blood volume as estimated by the erythrocyte count during the injection was not increased to any extent (table 1). While body weight may be used as a guide, I believe that an exact mathematical ratio cannot be given. The condition of the cardiovascular and renal systems must be considered as well as the clinical behavior of the patient during treatment.

HYDRATION FACTOR

During the treatment in my early cases I was in constant fear of producing a serious state of hydration of the brain. This fear dictated a type of treatment in which varying amounts of spinal fluid were drained both before and after the introduction of the hypotonic solution. Experience has shown that this procedure was incorrect according to the present conceptions.

At that time it seemed necessary to reduce the pressure of the cerebrospinal fluid by drainage until it approached atmospheric pressure, thus preventing any interference with drainage of fluid from the perivascular spaces.

A properly conducted forced perivascular drainage will allow withdrawal of as little spinal fluid as is necessary for laboratory procedure, allowing no more fluid to escape until after the beginning of the injection of hypotonic solution. The spinal fluid can then be allowed to drain and, on completion of the intravenous injection, the spinal needle should be immediately withdrawn. This procedure will largely prevent headache and vomiting; I believe that in this way one can control the only dangerous accident, cerebellar herniation.

The curative effect of this treatment seems to be due to certain fluid movements through the tissues of the central nervous system, movements not constant in all cases treated or during different periods in the same case. Possibly the production of a certain amount of hydration of the tissues of the central nervous system is therapeutic. In testing this factor a monkey with poliomyelitis was given eleven eight hour treatments, with four hour rest periods; 0.375 per cent sodium chloride was injected into the vein continuously at the rate of about 10 cc. per pound hour. During each treatment the subarachnoid space was closed except as 1 cc. of spinal fluid was removed at the beginning and the end of each period for cell study. This animal recovered without paralysis.

We have given thirty-three such treatments and during these periods the animals have seemed to be comfortable. They have never vomited and, except for short periods when they showed nervous irritability, they have eaten and behaved in a manner in nowise different from animals similarly treated with the spinal fluid draining.

I am cautious in using this factor in the treatment of human beings and have limited any closed period to half an hour. Spinal fluid pressure studies have shown considerable variation during the intravenous injection of 0.375 per cent sodium chloride. Apparently the production of a moderate amount of hydration of the brain and cord is in no way harmful, but before this principle is used to any extent in the treatment of human disease more research should be done.

THE EFFECT OF INTRAVENOUS HYPOTONIC SOLUTION ON INFLAMMATORY TISSUE

In 1932 I [7] was able to show that, when a hypotonic solution was injected intravenously, the fluid movement from the blood stream was greatest in inflamed tissues of the body. In cases of inflammation of the lungs or bronchi an acute edema of the lungs will develop almost as soon as the intravenous injection is begun. This has occurred in several cases of bronchitis associated with infections of the central nervous system and in one case of tuberculosis in a tabetic patient; also several times in monkeys with poliomyelitis that were suffering from pulmonary tuberculosis. In fact, two of these animals died of pulmonary edema.

TABLE 1.—*Case of Encephalitis in Baby Treated by Intravenous Drip in Which Moderate Generalized Edema Developed; Blood Volume Only Slightly Increased*

R. P., aged 4 months; weight, 14 pounds 12 ounces; Feb. 14, 1935. Cerebrospinal fluid: 7 cells, 6 lymphocytes, 1 polymorphonuclear leukocyte; chlorides 730 mg., red blood cells 4,700,000.

Time	Cumulative Totals Intravenous Drip, Cc.	Interval Amount Cerebrospinal Fluid, Cc.	Count of Cells	Red Blood Cells, Millions	Chlorides, Mg. per 100 Cc.	Sugar, Mg.	Temperature, F.	Pulse	Respiration	Urine, Cc.
10:35	.....	10	7	4.7	730	..	99.6	100	36	..
11:00	.....	..	..	4.1	...	..	....	...	..	5
11:35	230	6	4	...	850	66	99	100	36	..
12:10	.....	..	..	3.5	...	..	....	...	..	40
12:35	500	..	..	...	...	..	....	...	..	..
1:00	.....	..	..	3.9	...	..	99	140	36	..
1:35	750	2	17	...	...	..	99	140	36	..
2:00	.....	..	..	3.7	...	..	....	...	..	..
2:10	.....	..	..	...	...	..	....	...	..	120
2:35	1,110	22	11	...	760	66	....	...		(edema)
3:00	.....	..	..	3.6	...	..	....	...	..	..
3:10	.....	..	..	...	...	..	....	...	..	60
3:35	1,360	10	9	...	...	..	98	160	36	..
4:00	.....	..	..	4.2	770	..	....	(edema increased)		
4:35	1,589	20	2	...	720	..	....	...	..	..
5:00	1,850	7	10	4.43	...	..	....	...	..	75
6:00	2,080	10	..	4.43	...	..	98.2	146	36	..
7 hr. 25 min.	2,080	87								350

Solution 0.35 per cent sodium chloride given at average of 18.8 cc. per pound hour

In cases of pyuria a copious excretion of urine is found during treatment, with coincident lessening of the flow of spinal fluid. Hypodermic injections of pitressin in an effort to reduce the urinary output in these cases have achieved questionable success. In one human case and one in the monkey in which acute colitis was present, quantities of water were excreted from the colon. In the case of the monkey the excretion from the bowel nearly ceased in the interval between treatments, beginning again promptly on resumption of the intravenous injection. As a result, no spinal fluid was obtained during an eight hour treatment period (table 2). In one case of chorea in which there was an active endocarditis, two treatments were given without improvement; but a slight increase was noted in the patient's blood volume, which was checked at hourly intervals.

In one monkey with an inflammatory condition of the skin of the feet, large blisters were produced on the feet by the intravenous injection. These became progressively greater as the intravenous injection proceeded. Finally, during the active stage of poliomyelitis in the monkey we found the flow of spinal fluid greater than in the normal animal.

7. Retan, G. M.: Forced Spinal Drainage in Its Relation to Infections of the Central Nervous System, J. A. M. A. **99**: 826 (Sept. 3) 1932.

Inflammatory disease of the lungs, bronchi, kidneys or heart often contraindicates treatment. Inflammation of the colon will interfere with any clinical improvement that might otherwise be obtained.

CEREBELLAR HERNIATION

I believe that properly conducted forced perivascular drainage is a safe procedure and that cerebellar herniation, the only substantial danger, can be recognized early and prevented. In 130 treatments I have met this condition only twice.

My first encounter with cerebellar herniation occurred after I had been working with this method for two and a half years:

A girl, aged 9 years, developed chorea during convalescence from acute rheumatic fever and was treated ten days after its beginning. No breakfast or fluids were given; 30 cc. of spinal fluid was collected for some special laboratory tests, and 3,000 cc. of 0.45 per cent sodium chloride was injected during a four hour period. The patient vomited six times during this treatment. No occipital or suboccipital headache was complained of; the pulse rate continued around 120, respirations at 28, showing very little change during the treatment; in fact, the only untoward event was the vomiting.

TABLE 2.—*Experiment to Show Effect on Drainage of Spinal Fluid with Inflammatory Tissue Existing in Colon*

Monkey 39, weight 3.68 Kg., March 26, 1935. Inoculated 1 cc. 1:40 dilution Rockefeller Institute and Brodie mixed virus intercerebrally.

Time	Fluid Amount Intravenous Drip, Cc.	Cerebrospinal Fluid, Cc.	Cell Count Differential: Cells	Leukocytes, per Cent	Large Lymphocytes, per Cent	Mononuclears, per Cent	Polymorphonuclear Leukocytes, per Cent	Urine, Cc.	Med.
First Treatment (April 1, 1935)									
9:25 p.m.	Started								
9:45	puncture	...	501	72	19	8	0.5	12	1/9 p.
10:25	54	...	...	..	..	..	..	65	
11:25	91	0.2 (bl.)							
12:25 a.m.	76	1.4	183	89	..	4	7	8	
1:25	60	0.5		Diarrhea began				18	
3:25	83	0.4	116	89	2	6	3	24	
4:25	77	0		Watery discharge colon				27	1/9 p.
5:25	21	0	...	..	..	..	..	49	
8 hrs.	462	2.4						126	
Second Treatment (April 2, 1935)									
9:20 a.m.	Started	..	...	..	..	..	..	..	1/9 p.
10:20	100	0	...	..	..	..	..	44	1/9 p.
11:20	100	0	...	..	..	..	..	5	
12:20 p.m.	83	0	...	..	..	..	..	20	
1:20	105	0	...	..	..	..	..	15	
2:20	60	0	...	..	..	..	..	29	
3:20	62	0	...	..	..	..	..	9	1/9 p.
4:20	51	0	...	..	..	..	..	7	
5:20	37	0	...	..	..	..	..	9	
8 hrs.	598	0	...	..	..	..	..	138	

Solution 0.375 per cent sodium chloride

Fluid movement diverted from central nervous system to colon

During four-hour interval between treatments small amount of water excreted from the bowel at intervals of about one hour

Solution 0.375 per cent sodium chloride. Fur ruffled

Watery discharge from colon followed within five minutes of the beginning of the intravenous drip and continued through the eight-hour period of treatment. Note lack of cerebrospinal fluid production from the beginning of the diarrhea

After the completion of the intravenous injection the spinal fluid was allowed to drip for an hour and a half. Five minutes after the completion of the drainage severe generalized convulsions developed, with tremors over the whole body. The arms and legs were rigid. There was deep cyanosis. The temperature rose to 97 F., and the pulse was weak, irregular and rapid. The convulsions were controlled by ether inhalations, following which carbon dioxide and oxygen were given. The patient's condition was improved. The spinal fluid pres-

sure was found to be 4 mm. of mercury. Clonus could be easily produced at the ankles and fingers and seemed inexhaustible.

Four hours later there was a second, less severe, convulsion and a slight convulsion a half hour after this. The pulse was 126 and respirations were 28. During the next twelve hours she slept. Her color remained good. She voided several times involuntarily but at no time was there any complaint of suboccipital pain or any retraction of the neck. The next morning she ate her breakfast and seemed none the worse for her experience. The following day the choreiform movements had entirely ceased and her condition was excellent.

The objections to the procedure used in this case are as follows:

1. Food, and especially fluids, should not have been omitted before treatment was instituted.

2. Too much spinal fluid was removed before the intravenous injection was given.

3. The stylet should have been replaced in the needle for a short time on the advent of vomiting and perhaps for a second or third short period in case subsequent vomiting occurred.

4. The hour's drainage following the completion of the intravenous injection should not have been allowed.

The second case was one of bulbar poliomyelitis, complete paralysis of the throat of five days' duration, and partial paralysis of the tongue and of the legs. Examination showed 106 cells in the spinal fluid, of which 92 per cent were lymphocytes, at a pressure of 6 mm. of mercury, increasing with jugular compression. The patient was given 2,390 cc. of 0.45 per cent sodium chloride intravenously. A half hour after the treatment had begun his tongue protruded in the midline and continued to do so. Three hours after the beginning of the treatment the abdominal reflexes, which had been absent, returned but later were not obtained. His speech improved. Treatment was further uneventful. Following this, he was given a rectal drip of 500 cc. of physiologic solution of sodium chloride. Having shown considerable clinical improvement, he was given a similar treatment seventeen hours later. Ability to swallow fluids had not returned. He was given 1,300 cc. of 0.45 per cent sodium chloride. There was a complaint of severe suboccipital headache but no vomiting. The pulse rate and temperature were not disturbed.

At the completion of the intravenous injection the patient was given a rectal drip of physiologic solution of sodium chloride and the spinal fluid was allowed to drain for one and a half hours. One hour later he was cyanotic, a jerky type of respiration developed, the pulse was irregular, with a rate of 50 and of poor quality, and large coarse bubbling râles were heard in the chest. He was placed in a respirator and died in ten hours. Autopsy revealed a collar-shaped ridge in the cerebellum from contact with the posterior rim of the foramen magnum. The brain showed marked edema. It was the pathologist's opinion that herniation was not sufficient to cause death.

In this case a clysis of physiologic solution of sodium chloride should have preceded both treatments, and the period of drainage following the completion of the intravenous injection should not have been allowed. The interval between treatments should have been four or five hours, and 0.375 per cent would have been a better solution.

In the following outline the subject of cerebellar herniation is epitomized.

Incidence.—Frequent in septic meningitis.

Rare in other diseases; two in 130 treatments.

Has not occurred during intravenous hypotonic injection.

Symptoms and Signs.—Stage 1. Suboccipital pain and projectile vomiting followed by

Stage 2. Slowing of pulse, from 40 to 60, coincident with slowing of respiration; later, slow, jerky respiration followed by

Stage 3. Difficulty in swallowing: bloody spinal fluid, clonus, convulsions, respiratory paralysis.

Prevention and Treatment.—Stage 1. Insert stylet in lumbar needle for half hour. Continue intravenous injection. Symptoms will be relieved. Stylet is then removed. The intravenous injection should often be given at a more rapid rate.

Stage 2. Replace stylet at once or remove spinal needle. Discontinue intravenous drip. Elevate foot of bed.

Stage 3. Same treatment as for stage 2. If relief is not obtained, give intravenous injection of 50 per cent dextrose.

Admonition.—Do not remove a large amount of cerebrospinal fluid before beginning intravenous injection.

Do not drain cerebrospinal fluid after completion of intravenous injection.

This management will insure the patient against serious accident. These cases do not always follow the same pattern.

SEPTIC MENINGITIS

Forced perivascular drainage, in its present form at least, is not a cure for septic meningitis. I have treated twelve patients, all of whom have died. Since several did show considerable clinical improvement during the course of the treatment, it offered a certain modicum of courage to continue.

If the treatment is attempted, it should be instituted only when the case is seen early. There is certainly no hope for the later case. Furthermore, the organism must be seen in the direct smear of the spinal fluid; otherwise the operator may be dealing with an aseptic type of meningitis, secondary to an abscess contiguous to the dura. In these cases the treatment is distinctly contraindicated because of fear of converting an aseptic case into a septic one. There is a further difficulty, in the fact that cerebellar herniation is easily produced in this disease. In septic meningitis a plastic exudate attaches to the pia arachnoid, obstructing to some degree the normal flow of cerebrospinal fluid. If this fluid is obstructed in its passage, especially at the level of the foramen magnum, edema of the brain will be produced by the intravenous hypotonic solution. The brain stem is therefore forced into the foramen magnum. This accident can be prevented. It is discussed under the heading "cerebellar herniation." Unfortunately in the control of this complication it is necessary to interrupt the drainage of cerebrospinal fluid, and if this is done for any length of time the septic meningitis becomes worse.

Should an effective serum be available for the offending organism, it might be added to the hypotonic solution with the hope of carrying antibodies through the lesions.

SYDENHAM'S CHOREA

Chorea cases vary in their response to forced perivascular drainage. The most severe types of chorea have been the ones in which clinical improvement has been both prompt and complete. The mild types of hemichorea have been slow to improve and have required more vigorous treatment. I have treated eighteen cases; two of the milder type have been complete failures. One case showed definite improvement but was not cured after three treatments were given. Most of these cases were treated with 0.45 per cent sodium chloride. Two cases failed to improve with this solution but were promptly cured when 0.375 per cent sodium chloride was used.

Some have thought this treatment too radical for use in Sydenham's chorea. It certainly is indicated in severe cases and in cases which do not respond to other types of therapy. As to the question of radical procedure, forced drainage of the perivascular spaces can be so conducted as to be substantially safe.

POLIOMYELITIS

I have not yet had the opportunity to make a statistical study on which the evaluation of the treatment of acute human poliomyelitis must rest. The first case of preparalytic poliomyelitis treated by this method was treated by me in 1930.[8] I have since treated five other cases, three of which were preparalytic and two active bulbar involvement.

I believe that this disease is amenable to forced perivascular drainage and that with properly applied technic and procedure it can be definitely modified.

Experimental poliomyelitis in the monkey (Macacus rhesus) is in nearly every respect the same as the human disease. The two definite differences have to do with the virulence of the infection and the cell behavior of the cerebrospinal fluid. In the human disease the host is relatively resistant and one finds a fairly high percentage of recoveries without paralysis in untreated cases. Although the death rate varies in different epidemics, it is usually not high. There is, on the other hand, an entirely different picture with the experimental disease. When the virus is attenuated, a disease may be produced which does not kill all the animals; but with an adequate dose of virulent virus one can produce a disease in monkeys which causes complete skeletal paralysis and death in all infected animals. As far as I have been able to learn from a review of the literature, there have been no cures of this disease by any method when the monkey has been inoculated with adequate doses of virulent virus. It is therefore significant that six monkeys with acute poliomyelitis treated by this method have recovered without paralysis. A full report of this work will soon be published. Dr. O. D. Chapman has collaborated with me in this work.

A few of the important results will be mentioned here. 1. We have worked with both intranasal and intracerebral inoculations and report recoveries without paralysis following inoculation by both routes. 2. The concentration of the dose of virus does not seem to influence recovery. One of the animals that recovered without paralysis received intracerebrally fifty times the dose of virus which has killed all our control animals. In each experiment there has been one control animal similarly inoculated and untreated, in all of which complete skeletal paralysis has developed, followed by death. No animals have recovered when 0.45 per cent sodium chloride was used. 3. To date we have thought that our animals do best with eight hour periods of treatment followed by four hours of rest. One animal given fifty-nine hours of continuous treatment died. 4. We have had one recovery using the hydration factor in which no spinal fluid was allowed to drain with the exception of 1 cc. collected for cell study at the beginning and the end of each treatment. In this work our objective has been to test the various factors involved in the treatment in order to determine what factor or factors are essential. I believe that poliomyelitis, from its nature, is well adapted to this type of therapy. In this disease there is no acute inflammatory process existing in organs outside the central nervous system that will cause a diversion of fluid movement. The disease is related to the vascular system within the central nervous system. Here the nerve cell is richly supplied with blood, providing an opportunity for fluid to pass through these capillary walls and bathe the nerve cell. Perivascular infiltration, edema and hyperemia are present; at times there is petechial hemorrhage. The actual destruction of the nerve cell is a later event. This pathologic condition is found in the depths of the tissue, where it can be reached by this process of lavage. While the essential factor that explains the therapeutic results is not yet known, this type of therapy does profoundly modify acute poliomyelitis in the monkey.

My experience with the treatment of human poliomyelitis is insufficient for any conclusive statement. The four patients with preparalytic poliomyelitis recovered without paralysis or muscular weakness. It was significant, however, that both during and following the treatment the tremors and the nervous irritability of these patients was definitely relieved. Considered in relation to other experiences both in poliomyelitis and in other diseases, this fact may be of extreme importance.

A girl, aged 12 years, with bulbar paralysis, was treated. She had had fever, headache and vomiting for four days, inability to swallow and rapid breathing for three days, right facial paralysis, decreased movement of the diaphragm on both sides, more marked on the left, and for the past few hours increased difficulty in breathing. There was rigidity of the neck; the temperature was 103 and the cell count of the cerebrospinal fluid was 155. She was given three forced perivascular drainage treatments with 0.45 per cent sodium chloride. During the course of the first treatment her breathing definitely improved; following this treatment she was able to swallow for the first time in three days and drank milk and water. Six hours later she lost her ability to swallow. The temperature rose to 102. She was given a second similar treatment of 3 liters of 0.45 per cent sodium chloride. There was still further improvement in her breathing and she was again able to swallow for a period of seven hours. Following a third treatment she showed further improvement and for a third time regained her ability to swallow liquids. The respirations were stronger but the facial paralysis remained unchanged. From this point the patient made a steady gain. Facial paralysis was less obvious the following day; there was some weakness of the extensors of the left arm. Three days later she was given solid food for the first time and from this point she made a complete and uneventful recovery.

Viewed from our recent experiences the following criticism of the foregoing treatment should be made: First, the salt solution in the intravenous injections should have been 0.375 per cent instead of 0.45 per cent sodium chloride. Secondly, the interval between treatments should have been four hours rather than six or seven hours.

The technic that will produce the best results in the treatment of human poliomyelitis should be worked out by some one who thoroughly understands the subject and is conversant with the technical variations possible.

ENCEPHALITIS

Three cases of acute encephalitis have been treated. These were in infants with coma, severe continuous generalized convulsions, hyperpyrexia and various paralyses. The first case has already been reported.[8]

The second case was similar to the first and showed the same remarkable clinical response to treatment. This patient has since remained well.

The third patient, an infant, aged 1 year, with a temperature of 106, was in coma and had continuous convulsions, continuous convulsive movements of the eyeballs, periodic strabismus, transitory facial paralysis and a spastic paralysis of the right arm. The convulsions, coma and focal signs were all relieved by treatment. The temperature became normal. Unfortunately, a bronchial pneumonia developed which prevented further treatment, and he died.

8. Retan, G. M.: New York State J. Med. **31**: 1378 (Nov. 15) 1931.

One patient with chronic encephalitis of the parkinsonian type of four years' duration was given three treatments. He was almost completely relieved and remained so for a year, when he had a relapse. Forced perivascular drainage at that time was given without benefit. Fractions of spinal fluid from this patient gave the same cell picture that was found in the acute cases (table 3). I have treated several cases of chronic encephalitis of the parkinsonian type, of six and seven years' duration, without any clinical improvement. No cells were found in the spinal fluids.

Although experience is limited, the study of these cases suggests, first, that it is highly desirable to begin treatment of the more serious types of encephalitis as early as possible, with the hope of preventing as much cerebral damage as one can. Secondly, that the treatment should be pushed with vigor, and it would seem that eight hour treatments with four hour rest intervals would be the most successful, since these patients relapse in about twenty-four hours if only one treatment is given.

TABLE 3.—*Parkinsonian Type of Chronic Encephalitis of Four Years' Duration*

J. M., aged 27 years, Jan. 11, 1932.

Time	Cumulative Totals Intravenous Drip, Cc.	Interval Amount Cerebrospinal Fluid, Cc.	Cell Count Differential		
			Cells	Polymorphonuclear Leukocytes	Mononuclears
1:00	.....	18	8	0	8
2:00	350	90	29	0	29
3:00	690	32	12	3	9
4:00	1,000	37	11	0	11
5:00	.....	23	12	0	12
6:00	.....	26	11	0	11
5 hrs.	1,000	226			

Solution 0.45 per cent sodium chloride

Cell response: Cells mostly of mononuclear type. Lymphocytes and monocytes not differentiated. Similar cases of six and seven years' duration show no cell response. There was marked clinical improvement. Intravenous drip given too slowly and insufficient in amount; 1,200 cc. of water given by mouth during course of the treatment

The cell behavior in the cases treated both in acute encephalitis and in the one of four years' duration showed a similar pattern.

SYPHILIS OF THE CENTRAL NERVOUS SYSTEM

In 1930 I treated a case of syphilitic meningitis in an infant, aged 6 months. There was progressive hydrocephalus in spite of vigorous antisyphilitic treatment. The patient became steadily worse, and four forced perivascular drainage treatments were given. Arsenicals were added to the hypotonic solution with the hope of driving the arsenicals through the lesions. The patient made a remarkable recovery and has since developed normally. Following this I felt that a number of cases of syphilis of the central nervous system, treated at various stages of the disease, were benefited. It seemed that the best results were obtained in cases of tabes. Since this work was done before I had had the opportunity to improve technical procedures, I believe that there exists at the present time a fertile field for research in testing this method.

POSSIBLE DANGERS IN FORCED PERIVASCULAR DRAINAGE

As stated previously, the only substantial danger is that of possible cerebellar herniation.

Secondary infection into the subarachnoid space has been suggested but will not occur with aseptic technic, and the flow of the spinal fluid outward is also a protection against this accident.

In case of a dry tap caused by dehydration, repeated lumbar punctures should not be done until the hypotonic solution has been injected to the amount of about 500 cc. I have seen one extradural abscess produced by repeated dry taps.

The question of the possibility of producing a massive hemorrhage in cases in which petechial hemorrhage exists has been raised. This never occurs.

CONTRAINDICATIONS

Contraindications of the treatment are a cerebellar tumor or abscess or a closed cavity in the central nervous system from which cerebrospinal fluid cannot escape; inflammation of the lungs or bronchi; acute inflammatory disease of the heart, myocardial weakness or acute nephritis.

Acute colitis or pyuria does not contraindicate treatment but destroys its effectiveness.

PRESENT TECHNIC AND PROCEDURE

Three hours previous to treatment, the patient should be given food high in protein content and about three glasses of water. In case of dehydration a clysis of physiologic solution of sodium chloride should precede the drainage, after which lumbar puncture should be performed with the patient on a Bradford frame. The Queckenstedt sign is then tested. A specimen of spinal fluid is gathered for laboratory examination, care being exercised not to remove too much fluid. The stylet is then replaced in the needle and the needle gently rotated in order to enlarge the opening in the dura. The patient is rotated on the Bradford frame with the needle protruding through the window. An intravenous injection of hypotonic solution is given and, in case the patient is delirious or has a severe chorea, the internal saphenous vein is the vein of choice, for the reason that the foot can easily be immobilized to the frame. It is often desirable to tie the needle into the vein. When the flow of the intravenous solution has become established, the stylet is removed from the lumbar needle and the fluid allowed to drain. If the fluid spurts, the stylet should be introduced intermittently until a steady drop has been secured.

If frontal headache develops, an ice-cap or an injection of codeine may be used. If the headache is suboccipital or if it is followed by vomiting, the stylet should be inserted for about thirty minutes, after which the drainage can again proceed. Headache can also be controlled in many cases by increasing the flow of the hypotonic solution.

In case the flow of spinal fluid ceases altogether, a slight rotation of the needle upward will usually reestablish the flow. Should this occur early in the treatment and should the spinal drip not be reestablished after a period of thirty minutes, the intravenous injection should be stopped and a second lumbar puncture done. If it should occur late in the treatment, particularly following a free drainage of spinal fluid, it constitutes no reason for discontinuing the intravenous injection. Following the completion of the intravenous injection the spinal needle should be removed.

SUMMARY

1. Forced perivascular drainage is a powerful therapeutic agent.

2. Since it allows of considerable variation in procedure, it is still in an experimental stage of development.

3. Before its value can be known in the treatment of various diseases in which it may be indicated, statistical evidence is necessary.

4. In this discussion of the various factors involved, I have realized that future research may alter some of the present conceptions.

5. From my present experience I believe that forced perivascular drainage will be of use in the following infections of the central nervous system: acute poliomyelitis, acute and chronic encephalitis, chorea, syphilitic meningitis and tabes dorsalis.

6. While I am not in a position at the present time to advise the exact amount of hypotonic solution necessary in an individual case, 1 liter of hypotonic solution cannot be expected to be effective.

7. I believe that this treatment should not be used until the physician has thoroughly acquainted himself with the subject.

8. I further believe that this is a safe procedure and that the only danger is cerebellar herniation, which can be prevented and controlled.

9. If it can be shown that the hydration factor does not interfere with the therapeutic result, and if it can be shown that the method can be used with safety in the treatment of human beings, it will be an agent of the greatest value, since it will contribute to the comfort and safety of the patient.

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ABSTRACT OF DISCUSSION

DR. TEMPLE FAY, Philadelphia: Dr. Retan's clinical results are distinctly encouraging. The method employed in his treatment therefore entails important factors for consideration. That improvement has followed this method of treatment does not in itself indicate that the explanations for the improvement are at the present justified or that they may necessarily be due to so-called forced spinal drainage. I should like to ask Dr. Retan what the object of forced spinal drainage actually is in terms of relief to the central nervous system from certain pathologic states. Certainly the few cells washed out, as indicated in his studies, cannot be considered an index of clearing this perivascular space, supposedly occluded. If such were the case this method should have been attended with beneficial results in the suppurative meningitic group, all of which patients died in spite of continuous drainage and elaborate methods of hydration. Considering the amount of spinal fluid yielded by drainage in proportion to the fluid administered, as well as the relatively few cells "washed out," one cannot conclude that the method was responsible for the benefits, because in my experience with Sydenham's chorea with the parkinsonian syndrome and in a few cases of multiple sclerosis similar beneficial results have been obtained by forced drainage without permitting the patient to receive even the normal and usual intake of liquid. Encephalography produces a forced drainage in that all spinal fluid may be readjusted when an appropriate amount of air has been introduced to take its place. The beneficial effect on blood volume is that, as spinal fluid is withdrawn, an increase in blood volume within the craniovertebral cavity occurs to the limits of vascular distention, both as to the venous sinuses and as to the capillary-arterial bed. This new hyperemic state is maintained until spinal fluid returns to fill the cavity, and the advantage gained in blood volume cannot be disturbed unless there follows a subsequent period of hydration and an increase of intracranial pressure. I believe, therefore, that Dr. Retan's results may be attributed to an improved blood volume, better oxygenation and a better tissue function, in the same way that a chronic leg ulcer or bed sore responds to massage, increase in blood volume and better nutrition. Chloride values themselves may have a real part to play, but I doubt the value of the "washing out" process, which smacks of a laundry rather than a fundamental physical law relating to disturbances within a closed cavity, following the Monro-Kellie doctrine. The improved clinical results reported argue well for the method, but my own results and experience indicate that the damage of hydration is real and that the same results may be obtained by increasing the blood volume and associating this procedure with "dehydration," thus maintaining a more permanent shift in favor of better oxygen and nutrition.

DR. HENRY LOWENBURG, Philadelphia: The inherent hazards of a therapeutic procedure must always be compared with the inherent hazards of the disease to be treated. On this basis it is evident that in chorea this procedure may not become the treatment of choice. The severest cases of chorea have yielded to simple measures or to none at all. Shortened time of cure, while desirable, may not become the deciding factor in the presence of acknowledged risks. As Dr. Retan states, surgical drainage alone can never be expected to solve the problems of otitic and other forms of septic meningitis, because surgical drainage at its best is inadequate to drain and remove thick exudate and infected fluid. A single autopsy is sufficient to convince one of this. While all efforts, as this proposal, are to be encouraged, a great deal cannot be expected from them. Four recent fatalities from hemolytic streptococcus meningitis treated in this fashion with radical otitic surgery are sufficiently discouraging to hope for something better. In meningococcic meningitis, pathologic involvement is just as extensive. Cure results not from surgical drainage but from the use of specific antiserum or antitoxin. This is an eloquent example that the hope of the future for all other types of meningitis resides not in surgical drainage but in a potent antiserum, antitoxin, bacteriophage or diffusible chemical agent, which, though innocuous, is also powerfully antiseptic and sterilizing and possesses the ability to dissolve exudate.

DR. TRACY PUTNAM, Boston: Ever since Dr. Kubie's first description of the method of spinal drainage in 1927, we have occasionally used it in inflammatory diseases of the nervous system at the Neurological Unit of the City Hospital of Boston. We have begun to feel that it is a very valuable therapeutic agency, particularly in the milder forms of septic meningitis. If it is inadequate to cope with the more severe streptococcus and pneumococcus, this, I think, does not mean that it should be abandoned in the treatment of the milder types. We have had striking results in Bacillus coli and in staphylococcic meningitis with this method and also in draining compound fractures of the skull and spine. We have been somewhat less drastic than Dr. Retan and contented ourselves with producing a hydremia by forcing fluid by mouth. Dr. Retan's present work shows that intravenous injections of hypotonic solutions should be used. However, this method should be reserved for the definitely infectious diseases of the central nervous system, and little is to be expected from it in multiple sclerosis and the so-called encephalitides following infectious diseases, which, I am convinced, are not infectious in nature. If they are not infectious, if there is no specific toxin present, I think one cannot expect to accomplish much by the washing out process.

DR. GEORGE M. RETAN, Syracuse, N. Y.: The object of forced perivascular drainage is to wash products of inflammation, toxins, from the depth of inflamed areas in the central nervous system through the perivascular channels by changing the osmotic force of the blood stream. Of course, that is partly theoretical. Dr. Fay asks "Why perivascular drainage? How does one know that one is draining through the perivascular system?" Of course, the Virchow-Robins perivascular areas are drainage channels through which products of cell metabolism pass into the spinal fluid. If hypotonic injections are made into the blood stream of animals, a dilatation of these perivascular channels can be produced as well as of the pericellular fluid areas. The next support to this theory comes from the fact that cells are washed from perivascular areas during this treatment. Eighty-seven cells in the case mentioned may not be a large number, but the number of cells obtained in fractions of spinal fluid examined from various diseases has varied a great deal. In cases of chorea, one may obtain a small number, maybe seven or eight cells, as compared with one cell found in the original fluid. In acute encephalitis the cell response is definitely greater and fractions contain from twenty-five to 200 cells. Very frequently fractions are taken from as much as 90 cc. which has drained in one hour and which contains definitely more cells than were found in the original spinal

fluid. There is no constancy of fluid movements when massive injections of hypotonic solution are used. Some patients lose an enormous quantity of fluid by the kidney, almost as much as is given. Those patients, I believe, often do not obtain the therapeutic result that is desired. They do not do as well as patients who do not lose fluid from the kidney so rapidly. The state of hydration of the tissues of the body, when this treatment is begun, is of importance and for that reason I believe that one should fill as much as possible the tissues of the body with fluid in order to get a better clinical result. I have frequently treated purposely a patient four hours after the completion of the first treatment when the tissues of the body are pretty well choked with water, and I believe that a better clinical result is obtained in the second than in the first treatment. Much work remains in testing this hydration factor. I doubt whether forced perivascular drainage with the canal closed will ever be done. My object in studying the possible dangers of hydration and the therapeutic result is to determine the effect of a forced drainage treatment with the canal closed part of the time.

AN EPIDEMIC OF TRICHINOSIS IN MAINE

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This year marks the one hundredth anniversary of the naming of Trichina spiralis by Richard Owen, the material studied having been obtained in the dissecting room by Sir James Paget.[1] Trichinella spiralis was later suggested as a substitute by zoologists, because the name used by Owen had been applied five years before to a genus of Diptera. The same parasite seems to have been described by Tiedemann[2] in 1822, and the museum at Guy's Hospital contains a specimen of Trichinella prepared by Peacock in 1828. The anatomist Leidy[3] of Philadelphia first found trichinellae in pork in the year 1847. Zenker[4] described clinical trichinosis in 1860. The eosinophilia of trichinosis was observed by T. R. Brown,[5] then a student at Johns Hopkins, in 1897.

Osler[6] observed the parasites as a student at Trinity in 1868 and he has written that two years later in the dissecting room of the medical school at Toronto he found "159 cysts in a single drachm of one of the muscles of the arm" in the body of the erstwhile janitor of the hospital. In 1873 he[6a] saw a case of trichinosis in Traube's clinic at Berlin and wrote in his notebook: "So far as I can learn only four or five cases of trichinosis have occurred in Canada, one in Montreal, three in Hamilton, and two cases in which I discovered the parasite post mortem in Toronto. Other cases may and probably have occurred." In 1900 he[7] stated that "only rarely are cases diagnosed in hospital practice. With the exception of a typical case in one of Traube's wards (1873), I have never recognized an instance of the disease until the past eighteen months, during which time three cases have occurred in my service at the Johns Hopkins Hospital."

Only a little study is required to convince one that trichinosis is not a rare disease. According to Ransom's[2] 1915 report, U. S. government statistics then showed 1,550 cases with 240 deaths. During the year 1932, 263 cases were reported to the federal government from fifteen states. Conner[8] in 1929 reported fifty-two sporadic cases admitted to the two medical services at the New York Hospital during the preceding fifteen years. Spink and Augustine[9] have just described thirty-five unrelated cases seen in Boston during a three year period.[10]

The following epidemics have been recorded: fifty cases with four deaths in Boston in 1892;[11] fourteen cases in New York State in 1916;[12] forty-three cases in Spaniards in Albany, N. Y., in 1929;[13] fourteen cases in East St. Louis in 1930;[14] twenty-nine cases, all but one in Italians, in Pennsylvania in 1931;[2] a single family of Italians with eleven cases in 1932,[15] and forty-three Italians in Atlantic City in 1933.[16] A small epidemic caused by eating jerked bear meat was reported from California in 1932.[17] These figures are dwarfed by the German epidemics: Hedersleben with 337 cases,[18] Karlsruhe and Weingarten[19] in 1924 with 150 cases, and the polar bear epidemic at Stuttgart[20] in 1930 with eighty-eight cases and twelve fatalities.

A still more convincing means of estimating the frequency of trichinosis is from autopsy and dissecting room figures. In 1894 Thornbury[21] reported three cases found in twenty-one autopsies. Williams[22] found trichinellae in 5.34 per cent of 505 routine autopsies at Buffalo in 1901. Queen[23] at Rochester, N. Y., in 1931 studied the diaphragms by digestion methods and found 17.5 per cent positives in 344 autopsies; at Boston 27.6 per cent of fifty-eight autopsies were positive.[9] Riley and Scheifley[24] at Minneapolis in 1934 found 17.9 per cent of 117 cadavers showing encysted larvae; in a second series 20 per cent of fifty cadavers were positive.[9]

Man serves as an accidental host to Trichinella through the medium of meat from an infected animal, usually the hog. The gastric juice digests the wall of the encysted larvae, the parasites mature, copulation occurs, and the female deposits the embryos in the

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intestinal wall, whence, by means of the lymphatics, they gain the thoracic duct and the blood stream. The majority of the larvae reach the voluntary muscles, where they initiate an inflammatory reaction, which leads to their encystment. They have been found in many parts of the body. Their presence in the blood was first remarked by Herrick and Janeway [25] in 1909. In the year 1906 Frothingham [26] discovered trichinellae in the brain. Van Cott and Lintz [27] in 1914 found the parasite in the cerebrospinal fluid. Von Herrenschwand [28] in 1927 described large numbers of trichinellae in the external ocular muscles. Horlick and Bicknell [29] in 1929 found larvae in the heart muscle. In addition, larvae have been observed in the gallbladder bile,[29] in liver,[26] in a pleural effusion, in the milk of a lactating woman, in the pus from a furuncle,[11] in the pancreas, in the kidneys and in the intestinal wall.[29]

In the absence of an epidemic the variable symptoms of trichinosis may suggest other diseases. The diarrhea and vomiting have been designated food poisoning, a condition that is occasionally associated with trichinosis. The unexplained fever has been called influenza. Abdominal pain may lead one to suspect an inflammatory disease of the appendix, intestine or gallbladder, or a peptic ulcer.[8] Edema of the eyelids has suggested acute nephritis or frontal sinusitis.[30] The eye symptoms include photophobia, swelling of the conjunctiva and subconjunctival hemorrhages, so that the ophthalmologist may be the first physician to see the patient.[31] Diarrhea, somnolence and fever suggest typhoid;[8] this suspicion may be strengthened by the finding of typical rose spots or splenomegaly. Urticaria may be observed. A positive Kernig sign and absent knee reflexes have raised the question of meningitis.[32] Larvae have been demonstrated in the brain in cases of encephalitis.[33] Pain and tenderness in the muscles has been designated peripheral neuritis. Extreme hypotension may be the presenting sign.[34] Pneumonia and myocarditis are the most serious common complications. Marked muscle weakness, which lasts for many weeks or months into convalesence, is a not uncommon sequel and denotes a severe infestation.

REPORT OF OUTBREAK

Mrs. Anna F., an Italian housewife, aged 45, admitted to the Medical Service of the Maine General Hospital, Feb. 17, 1935, had become ill eleven days previously with epigastric pain, diarrhea and vomiting. The admission temperature was 100 F. and the blood pressure 70 systolic, 52 diastolic. She was lethargic and moderately dehydrated. No knee jerks were obtained. The urinalysis was negative except for 5 mg. of albumin. The blood Wassermann and Kahn tests were negative and the blood urea nitrogen was 17 mg. per hundred cubic centimeters. The leukocyte count was 17,500, with 84 per cent neutrophils and 3 per cent eosinophils. A surgical consultant found only mild tenderness in the gallbladder area and believed that it was not a surgical case. The vomiting disappeared but she continued to have several daily stools and complained of abdominal pain. The stool examination showed occult blood. Cultures of the urine, stools and blood were negative for the typhoid group.

February 23 the husband, Ralph F., was admitted. He had suddenly become ill with diarrhea and black stools one week before admission. The diarrhea had improved but he had grown weak, nervous and sleepless. The urine once showed a slight trace of albumin and once a few hyaline casts. The white blood count was 14,400, with 75 per cent neutrophils and 1 per cent eosinophils. He had a low grade fever. On the fourth hospital day, rose spots developed and the laboratory reported that cultures from the stool had yielded an organism resembling Bacillus typhosis. March 1, Widal tests on the blood of both husband and wife were positive in 1:80 dilution The following day it was found that Mrs. F. had a white blood count of 23,100 with 38.5 per cent eosinophils; Mr. F. showed 21,000 leukocytes with 44 per cent eosinophils. Mrs. F. seemed more toxic that afternoon, the next morning she presented signs of pneumonia in the right lower lobe, and on March 4 she died. At autopsy many trichinellae were found in the striated muscles.

Investigation of this family yielded the information that three of the seven children had been admitted to the Farrington Hospital with unexplained fever and swollen eyelids; all these patients were found to have eosinophilia and positive intradermal tests for trichinosis. Furthermore, Dominico Di D., an Italian, aged 40, had been admitted to the same institution on March 2 with fever, marked weakness, a positive Kernig sign, and absent knee reflexes. Lumbar puncture gave negative results. The leukocyte count was found to be 12,000, with 24 per cent eosinophils. The muscular weakness increased and it became impossible for him to swallow any but liquid food. Cough and respiratory embarrassment appeared and he was transferred to the Maine General Hospital, where he died with pulmonary edema. Autopsy showed a severe infestation with trichinellae and a focal leukocytic infiltration of the heart muscle.

The search for further cases was continued and a coherent story of the epidemic was obtained. Soon after New-Year's, Rocco A. had journeyed to the neighboring countryside and purchased two pigs. The animals were brought to his home in the city and were fed on corn and milk. Their recent rations had been garbage collected from a nearby FERA camp. January 19 they were slaughtered at the Portland Abattoir; one of the animals was sold on shares to two Italians, one of them our patient, Ralph F. Home-made sausage was manufactured from the latter animal. The sausages were allowed to dry and were widely distributed to friends. Their consumption was begun early in February; they were eaten raw or after very little cooking. Seventy-one persons are known to have eaten of the sausages and fifty-six individuals showed signs of infection with trichinosis.

The nine members of the F. family were all ill with the disease. Ralph F. was discharged from the hospital, in spite of marked muscle weakness, March 15. A month later he was readmitted because of a return of diarrhea, and trichinellae were found for the first time in his stools, two months after the onset of his illness. His strength has not been entirely regained in five months from the beginning of the disease. Nicholas M., a boy of 14, showed hypotension and marked weakness, so that no hand grips were obtained and he turned himself in bed with difficulty; he has not entirely recovered after five months. Specimens obtained by biopsy on Nicholas M. and Ralph F. showed leukocytic infiltration of the muscle, but no trichinellae were found. One patient showed urticaria while recovering from trichinosis.

Trichinellae were found in the pork, some of which had been used in the manufacture of the sausage. No parasites were discovered in the carcass of the remaining pig.

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The criteria used in determining positive cases were the clinical manifestations of trichinosis, eosinophilia, and a positive intradermal test. Twenty-six persons were definitely ill; all this group showed eosinophilia and positive skin tests, except the two fatal cases. The intradermal test was not attempted in the case of Mrs. Anna F. A test was applied to Dominico Di D., but through error it was not read for twenty-four hours. A total eosinophil count in excess of 500 per cubic millimeter was considered as constituting eosinophilia. Spink and Augustine[9] set 400 eosinophils as the upper limit of normal. This figure was arrived at by taking 10,000 as the upper limit of the normal leukocyte count and 5 per cent as the maximum of normal differential eosinophil count. Fifty-one individuals presented total eosinophil counts above this limit. As a rule, elevated white counts were found with high differential eosinophil counts; this was not invariably so, since one boy who was ill with trichinosis showed a leukocyte count of 9,400, with 51 per cent eosinophils. The highest differential eosinophil count was 74 per cent. The greatest total eosinophil count was 23,800; this represents a leukocyte count of 34,000, with 70 per cent eosinophils, and was the result of the first examination of the 2½ year old son of Ralph F. The child was afebrile and apparently well on the day of this test. Two days later he was admitted to the pediatric service of the Maine General Hospital with a cough and a rectal temperature of 104; the admission leukocyte count was 31,600 and the eosinophil count only 8 per cent, a drop in total eosinophil count from 23,800 to 2,528 per cubic millimeter. A rapidly falling eosinophil count has been held to indicate the presence of some serious complication. The original white count of Dominico Di D. was 12,000, with 24 per cent eosinophils. The day of his death the leukocyte count was 16,800 and the eosinophils had dropped to 1 per cent; this represents a fall in total eosinophil count from 2,880 to 168 per cubic millimeter. Nevertheless, Mrs. Ralph F. died with a total eosinophil count of 8,894 per cubic millimeter (23,100 leukocytes, with 38.5 per cent eosinophils).

Twenty-five of the seventy-one persons who had eaten the infected meat showed eosinophilia without signs or a history of illness. Since there is no natural immunity to trichinosis in man, the degree of infestation must determine the severity of the disease. It is believed that these cases represent latent infections, which were mild so far as disabling symptoms go. They should, however, show encysted trichinellae in their muscles.

COMMENT

In the past there has been no specific test for trichinosis, other than the actual demonstration of the parasites. Bachman[35] in 1927 described a precipitin test in infected laboratory animals and in 1928 an intradermal reaction, the antigen used being prepared from trichinellae larvae. Augustine and Theiler[36] in 1932 showed that both these tests were positive in hogs and human beings who had been infected with trichinosis; the skin reaction was found to be immediate rather than delayed, as is the case with guinea-pigs. The following year McCoy, Miller and Friedlander[37] reported that the intradermal reaction was positive in 90 per cent of eighty-eight known cases of trichinosis. All but one of the thirty-four cases of trichinosis tested by Spink and Augustine[9] gave positive skin tests. Several other observers[38] have described positive reactions in a small number of patients. Kilduffe,[39] after performing the skin test in thirty-three cases during an epidemic, concluded that the test possessed no advantage over eosinophil counts; but his conclusion is hardly fair, as pointed out by Friedlander,[40] since the reactions were not read at once.

The dried larva powder used as antigen for the skin tests in this epidemic was furnished by Dr. Benjamin Schwartz of the U. S. Department of Agriculture. Dilution was made with physiologic solution of sodium chloride to which had been added 0.25 per cent of tricresol; 0.1 cc. of a 1:10,000 solution was used. Skin tests were applied to sixty-eight individuals who had eaten the infected meat and the results were read in from five to ten minutes. All persons who had been definitely ill showed positive intradermal reactions. Two others were called questionably ill because of histories of sore throat and feverishness after eating the sausage. Both these patients were found to have subsequent eosinophilia but failed to react to the skin test on two occasions. They probably represent mild cases of trichinosis with negative skin reactions. There are twelve other persons who were not ill who displayed eosinophilia but did not show conclusive intradermal reactions. This means that half of the previously mentioned twenty-five persons with eosinophilia but no history of illness did give positive skin tests. Five persons who were not clinically ill and did not show eosinophilia at the time of examination are included in the fifty-six positive cases because of marked skin reactions.

Six hospital patients with various diseases were tested with the antigen as controls. Five failed to show an intradermal reaction; a child who was recovering from burns gave a strongly positive skin reaction both with the antigen and with the diluting fluid.

There is no specific in the treatment of trichinosis. Antimony and potassium tartrate has been suggested but has not stood the test of time.[41] In 1916 Salzer reported[42] that serum therapy cured trichinosis; his claims were disproved the next year by Schwartz.[42] Miller, McCoy and Bradford[43] treated infected laboratory animals with intravenous acriflavine, gentian violet, metaphen and iodine; they concluded that no concentration of any drug used was effective in killing the trichinellae without serious injury to the host. Catharsis appears to be useful in the initial diarrhea, but it is seldom possible to recognize trichinosis at this stage. Prophylaxis might be of great value; this consists in the proper cooking or refrigeration of pork, education of the public in the dangers of eating uncooked meat, and possibly in the use of the intradermal reaction in hogs or improvement in the examination of pork by means of the trichinoscope, which is in use in Germany.[44]

SUMMARY

1. The first epidemic of trichinosis to be recorded in the state of Maine, so far as we have been able to determine, is the largest epidemic to be reported in the United States.

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2. Infection in the present instance came from eating improperly cooked, home-made pork sausages; all persons, with a single exception, were Italians.

3. Trichinellae were found in the infected meat and in the striated muscles of the two fatal cases at autopsy.

4. Seventy-one individuals are known to have eaten the sausage, and fifty-six showed signs of infection.

5. Twenty-six persons were ill with probable trichinosis; satisfactory skin tests were applied to twenty-four of these patients and in all instances the intradermal reaction was positive. Two other individuals, who gave a history of infection of the upper respiratory tract, showed eosinophilia but gave negative skin tests.

6. Twenty-five of the seventy-one who had eaten meat from the diseased hog gave no history of illness but were found to have eosinophilia; twelve of this group reacted to the skin test and thirteen did not.

CONCLUSIONS

1. Individuals who eat small quantities of trichinous meat may present subsequent eosinophilia without definite illness; it is believed that these persons represent latent cases of trichinosis.

2. Bachman's intradermal reaction is positive in individuals who are or who have recently been ill with trichinosis; the test may give negative results in latent cases.

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STUDIES OF GALLBLADDER FUNCTION

XI: THE COMPOSITION OF THE GALLBLADDER BILE IN PREGNANCY

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The frequency with which gallbladder disease follows pregnancy [1] cannot be considered a mere coincidence but must be the result of some altered condition in the biliary system in pregnancy, which predisposes to gallstone formation. The relationship is well illustrated in the data of Fallon,[2] who in 1927 analyzed 1,100 operations on the gallbadder and bile ducts and found that 700 of the patients were women who had borne children.

We [3] have shown that alterations in the chemical composition of the bile accompany biliary tract disease. Our earlier studies demonstrated that damage to the gallbladder wall is accompanied by profound changes in the composition of the gallbladder bile.[4] Not only is this true of gallbladder damage but the liver bile may be altered in its composition by damage to the liver parenchyma.[3] One must, in addition, take into account the possibility that either gallbladder bile or liver bile may be altered in composition by factors primarily nonresident in the biliary tract, since gout, tuberculosis, diabetes, vitamin deficiency and thyroid disorders are frequently associated with gallstone disease.

Studies in this laboratory [5] and elsewhere [6] have shown that the majority of biliary calculi consist in large part of cholesterol. This fact, taken in conjunction with the hypercholesteremia nearly always found in the latter months of pregnancy,[7] would indicate that there may be a direct relationship between the two conditions. The opportunity presented itself of obtaining for study thirty-four specimens of gallbladder bile removed at term during cesarean section in patients with no previous history of gallbladder disease. Chemical analysis of this material forms the basis of the present report.

The Composition of Gallbladder Bile from Pregnant Women

Patient	Chloride, Milliequivalents per Liter	Calcium, Milliequivalents per Liter	Total Phosphorus, Mg. per 100 Cc.	Bile Salt, Mg. per 100 Cc.	Cholesterol, Mg. per 100 Cc.	Ratio of Bile Salt to Cholesterol
Mo.	11.8	31	195	3,610	1,000	3.6
Fl.	17.5	15	89	3,610	616	5.8
Co.	18.0	15	180	4,400	...	...
Ba.	18.2	18	137	4,690	...	...
Pa.	30.6	11	114	3,810	482	7.9
Cl.	30.8	17	140	2,920	580	5.0
Wa.	32.4	12	84	2,000	330	6.0
Bu.	33.5	14	114	4,000	540	7.4
Pl.	35.0	11	94	3,600	404	8.9
Ha.	36.9	12	96	2,320	201	11.5
Mu.	37.3	11	92	2,250	396	5.7
DeL.	39.4	21	106	2,360	272	8.6
Ho.	40.8	12	107	3,120	285	10.9
Tu.	44.1	..	...	2,280	343	6.6
Ga.	45.7	9	80	2,280	276	8.2
Br.	46.3	12	92	1,640	...	...
Ro.	47.5	18	101	2,580	520	4.9
Ec.	50.0	16	80	2,000	357	5.6
Ka.	50.8	11	110	2,280	533	4.2
Wi.	50.8	18	103	1,980	245	8.0
Vo.	52.8	14	92	2,280	490	4.6
Do.	54.7	6	77	4,400	422	10.4
Ho.	56.1	11	98	2,080	400	5.2
Ho.	57.5	5	..	1,960	...	...
Di.	57.5	10	80	3,600	400	9.0
Do.	61.2	6	34	1,000	130	7.6
Sw.	61.2	7	66	2,980	328	9.0
Fe.	61.8	7	59	2,000	278	7.9
Mi.	64.3	9	57	2,220	181	12.2
Ta.	70.6	8	69	2,860	276	10.3
Wi.	73.8	18	54	1,520	220	6.9
Li.	80.8	8	34	1,430	160	8.9
Pa.	....	..	89	1,900	410	4.6
We.	....	..	..	2,300	220	10.4

RESULTS

In every instance the gallbladder wall was reported as normal in appearance, although the viscus was as a rule found to be distended at operation. The bile was analyzed for chloride, calcium, bile salt and cholesterol by the methods reported in our earlier papers. The total phosphorus was determined by the method of the Youngburgs [8] after digestion by the method of McCay.[9]

In the accompanying table the data are arranged according to the chloride concentrations. A few of the specimens show a chloride concentration within the range of normal gallbladder bile, while in the remaining specimens the chloride concentration is similar to the mean concentration found in the bile from a diseased noncalculous gallbladder. The calcium concentration varied from 5 to 31 milliequivalents per liter. The

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lowest concentration is approximately the same as that for hepatic bile, while the higher figures are similar to the concentrations of calcium in normal gallbladder bile. The mean concentration is similar to that found in patients with chronic noncalculous cholecystitis.[5] Total phosphorus varied from 34 to 195 milliequivalents per liter.

The bile salt concentration, expressed as sodium cholate, varied from 1,000 to 4,690 mg. per hundred cubic centimeters. In every instance the concentration of bile salt was below the concentration found in normal gallbladder bile, the highest concentration in the pregnancy group being about one-half the concentration that we have found to be present in normal gallbladder bile.

The cholesterol concentrations varied from 130 to 1,000 mg. per hundred cubic centimeters. All but three were above 200, and eighteen of the thirty were above 300. Ten specimens were analyzed by a digitonin method[10] for the presence of cholesterol esters, and in each instance no ester was present.

COMMENT

In an earlier paper of this series we reported on the chloride, calcium and bile salt concentrations of normal human gallbladder bile obtained from two living patients.[4] They showed a close similarity to the normal gallbladder bile of the dog. Because of the small quantity of bile available it was not possible to make cholesterol analyses.

The data here presented represent the only analyses of gallbladder bile from pregnant women in which the bile was obtained during life.

Only a few data are available in the literature on the concentration of cholesterol in normal human gallbladder bile. Much of the material termed normal cannot be so considered.[11] The only figures that approximate the normal are those given by Fowweather and Collinson[6] on bile from patients who died accidental deaths and from whom bile was obtained within three to four hours after death. The values given by them range from 200 to 280 mg. per hundred cubic centimeters, with a mean of 230 mg. Frerichs (cited by Hammarsten[12]) gives two analyses on bile from patients who died from accidents, the values being 160 and 260 mg. per hundred cubic centimeters. Four analyses for bile salt concentration in supposedly normal gallbladder bile (cited by Hammarsten) are 7,220, 9,140, 10,790 and 5,660 mg. per hundred cubic centimeters.

In general, the specimens of bile that we obtained from pregnant women were higher in the chloride and cholesterol concentrations and lower in the calcium and bile salt concentrations than is normal gallbladder bile.

The high values found for cholesterol in the majority of instances, which confirm the high values found post mortem by McNee, indicate either that the liver bile in pregnancy contains an increased concentration of cholesterol or that cholesterol is added to the bile while it is in the gallbladder. Several of the highest cholesterol values were found in specimens showing the closest approach to normal gallbladder bile with regard to the chloride and calcium concentration. This would seem to indicate that the hepatic bile entering the gallbladder must be abnormally high in cholesterol.

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The high bile cholesterol also indicates that hypercholesteremia is not, as Bacmeister and Havers[13] suggested, due to the retention of cholesterol in the blood as a result of a decreased output in the bile but indicates rather that the cholesterol in the blood is higher because of overproduction. An interesting feature of this aspect of the problem is that, although the cholesterol of the normal blood consists of approximately 40 per cent esters and 60 per cent free cholesterol, a relationship which, according to Boyd,[7] is not disturbed in pregnancy, ten specimens of bile analyzed by us for ester content contained no esters.

The high chloride and low calcium values found in a number of the specimens indicate that there is often in pregnancy a disturbance in the absorptive function of the gallbladder, since the normal gallbladder removes chloride and concentrates calcium.

The low bile salt concentrations may be due to several factors. Since bile salts are synthesized in the liver, any impairment of liver function may result in a decrease in bile salt production. The data indicating an impairment of functional activity of the gallbladder wall would also suggest that the decrease in bile salt concentration may at least in part be due to increased absorption of the bile salt such as occurs when the gallbladder wall is damaged.

The bile salt-cholesterol ratio was found to vary widely, but in every instance the ratio was below the normal as given by Newman.[14] The data as a whole show a wide spread in the results of analysis for the various constituents studied. They indicate that in pregnancy the composition of the liver bile is altered in respect to the cholesterol and bile salt concentrations. The data on chloride and calcium strongly suggest, in view of our earlier studies, that the gallbladder wall is affected so that it cannot remove chloride and concentrate calcium to the same degree as previously. The low bile salt-cholesterol ratios are in the direction of those found in the more severely damaged calculous gallbladders.

CONCLUSIONS

Data from the study of thirty-four specimens of gallbladder bile removed from living women at term indicate that in pregnancy both the composition of the liver bile and the absorptive function of the gallbladder are changed from the normal. In general, the cholesterol concentration of the gallbladder bile is increased. In every instance the bile salt concentration is below the normal for gallbladder bile. The changes in these two constituents in pregnancy bile are in the direction that one would expect in the precursor stage of calculus formation.

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Energy in One Gram of Protein.—Within fairly wide limits, proteins are interchangeable with carbohydrates as a source of energy in the sense that 1 gram of either yields 4 calories when burned in the body; but it is not good physiological or pecuniary economy to use too much protein merely as fuel, for this involves, as has been independently emphasized by Chittenden and by Folin, the constant overcharging of the body fluids with waste nitrogenous matter. Good judgment neither makes a fad of seeking a minimum protein intake nor attempts to rationalize among sedentary people the highly carnivorous habits which may be natural and harmless under frontier conditions.—Sherman, H. C.: Food and Health, New York, Macmillan Company, 1934.

Clinical Notes, Suggestions and New Instruments

PREVENTION OF SKIN DIGESTION IN HIGH INTESTINAL FISTULAS

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The treatment of high intestinal fistulas is a serious problem because of the interference with fluid and mineral balance and because the distressing digestion of the wound margin and skin surface delays healing, interferes with the patient's rest and causes him to refrain from eating. He soon learns that ingestion of food is followed by secretion of digestive juices, which reach the abdominal surface and cause intolerable distress. The problem to be discussed here is not the replacement of the lost fluid and solutes, although that may be vital in cases in which the loss is almost complete.[1]

Efforts to control the digestion of the abdominal wall have been only partly successful. They have been based on three main principles:[2] (1) the chemical neutralization of tryptic ferments by combining them with an excess of peptone powder, protein and fat;[3] (2) adsorption of the ferments by finely divided charcoal or kaolin,[4] and (3) removal of the ferments from the fistula by continuous irrigation[5] or continuous suction.[6] All these methods are ingenious and, to a varying extent, useful, but even in careful hands any of them will allow some contact of ferment and skin and thus permit further distress to the patient and destruction of the abdominal wall. Attempts to protect the skin by waterproofing with petrolatum and ointments have done more harm than good, because the drainage seeped between skin and ointment where it could not be interfered with by the efforts at chemical protection.

In a number of recent cases of high intestinal fistula in which skin digestion was an important factor, I have been able to control it by coating the skin with a thin film of rubber. Properly applied, and applied early enough, latex will adhere to the skin surface and will not allow ferments to seep beneath it. It will not interfere with the simultaneous use of chemical combination or adsorption or suction to prevent the ferments from acting within the sinus. The film is soothing and protecting to the skin and is comfortable and pliable. It does not require replacement except at infrequent intervals.

Latex is the juice of the rubber tree. It is a milky substance with an alkaline reaction. It is readily obtained and is inexpensive. It dries readily and when dried on the skin leaves adherent to the skin a transparent film of rubber. If the solution is acidified with dilute acetic acid the rubber will promptly precipitate out of it as a similar film. (One of the manufacturers of surgical supplies sells under a trade name small collapsible tubes of a substance resembling latex, which leaves a similar film of rubber and can be used in the same way.)

Care must be taken that the application is made on a clean dry surface, or it will not adhere. It should stop at the margin of the fistula and should be made early enough so that skin digestion has not rendered the entire surface raw and weeping. In recent cases I have applied the latex as a prophylactic dressing to the skin when I anticipated the possibility of trypsin reaching it instead of waiting for the digestion to occur. The film of latex must be spread thinly with a wooden applicator, so that it will dry readily, or else be precipitated with an acid wash. If a gauze dressing is to be applied over it, the rubber surface should be powdered. I have ordinarily used this technic combined with the heaped up kaolin dressing described by CoTui,[4] in which case the kaolin serves as a powder to prevent the rubber from adhering to the outer gauze dressing. Patients are very comfortable with this dressing, and repair of the fistula is rapid.

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5. Erdman.[1] Cheever, David: Concerning Traumatic Rupture of the Duodenum and Duodenal Fistula, Boston M. & S. J. **173**: 454 (Sept. 23) 1915.
6. Lahey, F. H.: Treatment of Duodenal Fistula by Suction, S. Clin. North America **4**: 1489 (Dec.) 1924.

Council on Physical Therapy

THE COUNCIL ON PHYSICAL THERAPY HAS ADOPTED THE FOLLOWING REPORT.

HOWARD A. CARTER, Secretary.

BARR ULTRA-SHORT WAVE RADIOTHERMY UNIT MODEL SW-5 ACCEPTABLE

Manufacturer: Barr Laboratories, Inc., New York.

This unit is recommended by the manufacturer for medical and surgical diathermy. It includes attachments for tissue cutting, coagulation and desiccation; all are of a monopolar type.

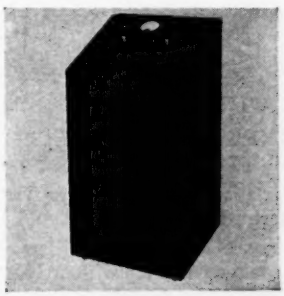

Fig. 1.—Barr Ultra-Short Wave Radiothermy Unit Model SW-5.

The standard machine is a one-tube, self-excited oscillator designed to oscillate at 9 meters, but the firm is prepared to offer a unit of any wavelength from 4 to 30 meters. The input power is about 680 watts. Since there is no acceptable method for measuring the output power of diathermy machines, this value is not stated. The shipping weight is about 175 pounds. Operating the unit under full load for two hours indicated that the temperature rise of the transformer and of the cabinet was within the limits of safety established by the Council. (Figure 2 is a schematic diagram of the circuit).

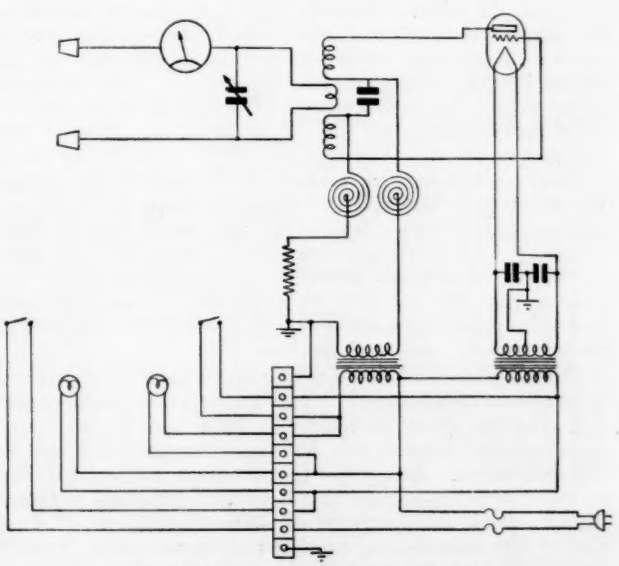

Fig. 2.—Schematic diagram of the circuit.

The tissue heating ability of the machine was investigated in a clinic acceptable to the Council. Cuff electrodes, about 2.5 cm. in diameter and about 50 cm. long, were used. When applied to the patient, the cuffs were separated from the skin by about one inch of felt. Thermocouples were introduced into the subcutaneous and deep-lying tissues (quadriceps extensor) of the human thigh. While the machine was being operated at the patient's tolerance, the temperature rise (average

of eight tests) observed at the beginning and end of twenty minute periods, the thermocouples being removed during the time of treatment. According to the results submitted, the temperature rise of the deep-lying tissues of the thigh was higher than that obtained with conventional diathermy—the criterion for evaluating short wave machines which the Council had adopted. The Council's investigator reported that the machine supplied sufficient energy to heat the body tissues whenever such treatment is indicated and gave satisfactory service for tissue cutting.

Burns may be produced by this machine but, with ordinary care, they may be avoided; it is highly probable that they will not occur and the danger is much less than with conventional diathermy.

In view of the foregoing report, the Council on Physical Therapy voted to include the Barr Ultra-Short Wave Radiothermy Unit Model SW-5 in its list of accepted apparatus.

Committee on Foods

THE COMMITTEE HAS AUTHORIZED PUBLICATION OF THE FOLLOWING REPORT.

RAYMOND HERTWIG, Secretary.

NOT ACCEPTABLE

1. LOEB'S GLUTEN FLOUR
2. FARWELL & RHINES CRISS CROSS BRAND GLUTEN FLOUR

Manufacturers.—(1) Loeb's Dietetic Food Co., New York. (2) Farwell & Rhines Co., Watertown, N. Y.

Description.—Gluten flour prepared from wheat flour by removal of part of the starch, containing not more than 10 per cent moisture and, calculated on the water-free basis, not less than 7.1 per cent nitrogen nor more than 56 per cent nitrogen-free extract (the protein factor 5.7 being used) and not more than 44 per cent of starch (as determined by the diastase method).

Uselessness as a Special Purpose Food.—These gluten flours are manufactured specially for use in diets restricted in dextrose formers. To be eligible for acceptance, such type of flour shall contain dextrose formers yielding dextrose in an amount not greater than 3.3 Gm. per hundred cubic centimeters (the dextrose equivalence being computed as the carbohydrate, plus 58 per cent of the protein, plus 10 per cent of the fat content of the food). Dextrose formers of these gluten flours, however, yield approximately 50 Gm. of dextrose per hundred cubic centimeters.

There is authoritative evidence that commercially prepared special diabetic foods are of limited usefulness to the diabetic patient and that the availability of insulin makes them no longer necessary. Artificial substitutes for ordinary foods are not to be favored; it is much better for the diabetic patient to learn how to plan his diet with foods in common use and readily available. The diet should be exactly prescribed in carbohydrate, protein and fat, and total calories.

The designation of a food as a "diabetic food" merely because it is low in carbohydrates is now unwarranted and misleading and gives the erroneous impression either that the food taken in unrestricted quantities in diabetes is harmless or that it has remedial action. Except for the necessity of restricting foods to avoid overstepping the food tolerance, there are no special diabetic nutritional requirements. The exploitation of starch-free or low carbohydrate foods containing an excess of protein for use by diabetic patients is unwarranted. Protein may be tolerated almost as poorly, if not quite as poorly, as starch in diabetes.

Because Loeb's Gluten Flour and Farwell & Rhines Criss Cross Brand Gluten Flour are adjudged without usefulness or special adaptability for inclusion in diets restricted in dextrose formers, they will not be listed among the accepted foods of the Committee on Foods.

Gluten flours of other manufacturers, for the reasons stated, are not eligible for acceptance.

ACCEPTED FOODS

THE FOLLOWING PRODUCTS HAVE BEEN ACCEPTED BY THE COMMITTEE ON FOODS OF THE AMERICAN MEDICAL ASSOCIATION FOLLOWING ANY NECESSARY CORRECTIONS OF THE LABELS AND ADVERTISING TO CONFORM TO THE RULES AND REGULATIONS. THESE PRODUCTS ARE APPROVED FOR ADVERTISING IN THE PUBLICATIONS OF THE AMERICAN MEDICAL ASSOCIATION, AND FOR GENERAL PROMULGATION TO THE PUBLIC. THEY WILL BE INCLUDED IN THE BOOK OF ACCEPTED FOODS TO BE PUBLISHED BY THE AMERICAN MEDICAL ASSOCIATION.

RAYMOND HERTWIG, Secretary.

1. MARY'S BEST BRAND GOLDEN TABLE SYRUP
2. SHURFINE BRAND GOLDEN SYRUP
3. SHURFINE BRAND WHITE SYRUP

Distributors.—1. Domanik Wholesale Grocery Company, Racine, Wis. 2 and 3. National Retailer Owned Grocers, Chicago.

Packer.—D. B. Scully Syrup Company, Chicago.

Description.—1 and 2. Table syrup; corn syrup flavored with refiners' syrup. The same as Banner Blue Syrup (THE JOURNAL, March 5, 1932, p. 817).

3. Table syrup; corn syrup sweetened with rock candy syrup; flavored with vanillin and coumarin. The same as White Crystal Table Syrup (THE JOURNAL, April 15, 1933, p. 1174).

MELLOW BRAND MILK—HOMOGENIZED, PASTEURIZED

Distributor.—Brown's Creamery Co., Detroit.

Description.—Bottled, pasteurized, homogenized milk.

Preparation.—Milk obtained from tuberculin tested herds, government and company inspected, is pasteurized by the holding method (61 C. for thirty minutes), homogenized, cooled and bottled, by the usual procedure (THE JOURNAL, Sept. 1, 1934, p. 681).

Analysis.—Standardized to contain not less than 3.5 per cent of milk fat.

Calories.—0.7 per gram; 20 per ounce.

Claims of Distributor.—The cream does not separate. The curd formed in the stomach is softer than that from unhomogenized milk.

RED & WHITE BRAND UNSWEETENED HAWAIIAN PINEAPPLE JUICE

Distributor.—Red & White Corporation, San Francisco.

Packer.—Alexander & Baldwin, Ltd., San Francisco.

Description.—Canned unsweetened pineapple juice retaining in high degree the natural vitamin C content. The same as Greetings Brand Pineapple Juice (Unsweetened) (THE JOURNAL, Aug. 17, 1935, p. 513).

OUR LADY'S BRAND MUSHROOMS BUTTONS (FANCY—FIRST CHOICE—MINIATURE)—SLICED—SLICED STEMS AND PIECES

Packer.—Michigan Mushroom Co., Niles, Mich.

Description.—Cooked mushrooms, whole, sliced, or sliced pieces and stems, with added water and salt; the same as Excelsa Mushrooms, Fancy Buttons, Pieces and Stems, and Sliced (THE JOURNAL, Aug. 4, 1934, p. 342).

PURINA BRAND WHOLE WHEAT BREAD

Manufacturer.—Franke's Hot Shops, Inc., Little Rock, Ark.

Description.—A whole wheat bread made by the straight dough method (method described in THE JOURNAL, March 12, 1932, p. 889); prepared from whole wheat flour, water, powdered skim milk, sugar, hydrogenated cottonseed oil, yeast, salt, malt extract and a yeast food containing calcium sulphate, ammonium chloride, sodium chloride and potassium bromate.

PHYSICIANS SPECIALIZING IN PATHOLOGY AND CLINICAL PATHOLOGY

PREPARED BY THE COUNCIL ON MEDICAL EDUCATION AND HOSPITALS

The Council on Medical Education and Hospitals has maintained lists of approved clinical laboratories and physicians specializing in pathology and clinical laboratory work since 1926. The list of approved clinical laboratories was discontinued in 1931 and replaced by the list of physicians specializing in pathology and clinical pathology.

The Council has always held that the practice of pathology and clinical pathology is a part of the practice of medicine. In many states the law does not specifically prohibit the interpretation of laboratory observations by others than licensed physicians, so that laymen have entered upon this field of practice. Through the activities of non-medical clinical laboratories the interests of the patient are jeopardized and frequently the true source, nature and progress of the disease are not ascertained. There is satisfactory evidence of the salutary influence of the Council on Medical Education and Hospitals in the gradual decrease in the number of lay-supervised hospital and independent clinical laboratories. Physicians everywhere have been constantly reminded of the importance of the medical laboratory and have been encouraged to patronize only such clinical laboratories as are directed by physicians listed by the Council or eligible for such recognition.

After nine years of experience with the clinical laboratory situation, results, no less satisfactory than the supervision established over medical schools and hospitals, are being realized through the Council's endeavors.

The following list contains the names of 852 physicians who were found to meet the "Essentials" and were recommended by the Council's advisers. Those engaged in teaching, research and other activities are admitted, as well as those in active practice. For the list of pathologists in government service, see page 1351.

LIST OF PATHOLOGISTS

NAME	ADDRESS
ALABAMA	
Birmingham	
Graham, Geo. S.	1023 S. 20th St.
Fairfield	
Jones, Walter C.	Tennessee Coal, Iron and Railroad Hospital
Montgomery	
Trumper, Abraham	201 Montgomery St.
ARIZONA	
Phoenix	
Hartgraves, Thos. A.	10th St. and McDowell Rd.
Mills, H. P.	15 E. Monroe St.
Tucson	
Hicks, Robert Alan	1800 E. Speedway
ARKANSAS	
Hot Springs	
Lee, Dee C.	236 Central Ave.
Little Rock	
Hoge, S. F.	215 E. 6th St.
Thatcher, Harvey S.	300 W. Markham St.
Pine Bluff	
Pittman, Wm. G.	202 Pine St.
CALIFORNIA	
Berkeley	
Reich, Wm. W.	2236 E. Durant Ave.
Hollywood	
Andrews, Vernon L.	1322 N. Vermont Ave.
Loma Linda	
Cutler, O. I.	
Long Beach	
Mikels, Benjamin M.	Seaside Hospital
Shackford, B. C.	117 E. 8th St.
Los Angeles	
Bettin, Mona E.	727 W. 7th St.
Bonynge, Chas. W.	1930 Wilshire Blvd.
Brem, Walter V.	657 S. Westlake Ave.
Butt, Edward M.	1930 Wilshire Blvd.
Evans, Newton	1200 N. State St.
Hall, Ernest M.	3551 University Ave.
Hammack, Roy W.	657 S. Westlake Ave.
Hemken, Louisa	3551 University Ave.
Hill, Robt. B.	511 S. Bonnie Brae
Hyland, Clarence M.	4614 Sunset Blvd.
Kimball, Theodore S.	1200 N. State St.
Lindberg, Ludwig	1407 S. Hope St.
Maner, Geo. D.	657 S. Westlake Ave
Pratt, Orlyn B.	312 N. Boyle Ave
Setzler, Geo. B.	1052 W. 6th St.
Zeiler, A. H.	657 S. Westlake Ave.
Oakland	
Glenn, Robt. A.	Samuel Merritt Hospital
Michael, Paul	434, 30th St.
Moore, Gertrude	2404 Broadway
Olive View	
Bogen, Emil	Olive View Sanatorium
Pasadena	
Foord, Alvin G.	Pasadena Hospital
Sturdivant, B. Frank	St. Luke's Hospital
Pomona	
Case, Lucius W.	1798 N. Garey St.
Johnson, Barton W.	353 N. Gibbs St.
Redlands	
Taltavall, Wm. A.	47 E. Vine St.
Sacramento	
Christman, Paul Wm.	1027, 10th St.
Guttmann, Paul H.	1127, 11th St.
San Diego	
Ball, Howard A.	San Diego Co. Gen. Hosp.
Elliott, Frances P.	233 A St.
Pickard, Rawson J.	520 E St.
Sumerlin, Harold S.	2001, 4th Ave.
Thompson, Harold A.	233 A St.
San Francisco	
Bolin, Zera E.	490 Post St.
Carr, Jesse L.	51 San Andreas Way
Lippman, Marion H.	135 Stockton St.
McNaught, James B.	2398 Sacramento St.
Oliver, Harry R.	490 Post St.
Perry, Isabella Hester	Univ. of Calif. Med. Sch.
Smith, Elmer W.	2200 Hayes St.
Smith, Pearl M.	3700 California St.
Stowe, W. Parker	St. Luke's Hospital
Victors, Ernst A.	450 Sutter St.
Wood, David A.	2398 Sacramento St.
Wyckoff, Harry A.	Stanford Univ. Hosp.
San Jose	
Campbell, Lenore D.	990 E. Santa Clara St.
Santa Ana	
Martell, B. S.	115 Owens Dr.
Santa Monica	
Kosky, Alfred A.	1250, 16th St.
McLean, Wm. J.	958, 24th St.
Stockton	
Holliger, Chas. D.	242 N. Sutter St.
COLORADO	
Colorado Springs	
Ryder, Charles T.	1626 Wood Ave.
Staines, M. Ethelyn	23 E. Pikes Peak Ave.
Denver	
Black, William C.	4200 E. 9th Ave.
Carson, Paul C.	Presbyterian Hospital
Dobos, E. I.	1818 Humboldt St.
Freshman, A. W.	227, 16th St.
Hillkowitz, Philip	227, 16th St.
Jones, Rodney H.	227, 16th St.
Kingry, Charles B.	1616 Tremont Pl.
Mugrage, Edward R.	4200 E. 9th Ave.
Queen, Frank B.	4200 E. 9th Ave.
Thorsness, Edwin T.	Denver General Hosp.
Williams, George Z.	227, 16th St.
Williams, Wm. W.	209, 16th St.
Pueblo	
Dunlop, J. N.	Corwin Hospital
Maynard, C. W.	702 N. Main St.
CONNECTICUT	
Hartford	
Allen, Wilmar M.	20 S. Hudson St.
Hastings, Louis P.	370 Collins St.
Kendall, Ralph E.	20 S. Hudson St.
Middletown	
Beauchemin, Joseph A.	Connecticut State Hosp.
Fisher, Jessie W.	28 Crescent St.
New Haven	
Bartlett, Chas. J.	Grace Hospital
Norwalk	
Murray, Archibald.	Norwalk General Hospital
Stamford	
Weaver, Bruce S.	77 South St.
Waterbury	
Collins, Joseph O.	64 Robbins St
DELAWARE	
Wilmington	
Gay, Douglas M.	Delaware Hospital
DISTRICT OF COLUMBIA	
Washington	
Cajigas, Tomas	1801 Eye St., N.W.
Choisser, Roger Morrison	1335 H St., N.W.
Dardinski, Vincent J.	Georgetown Univ. School of Medicine
Keilty, Robert A.	1801 Eye St., N.W.
Langenstrass, K. H.	St. Elizabeths Hospital
Lindsay, Janvier W.	1726 Eye St., N.W.
Neuman, Lester	1835 Eye St., N.W.
Rice, E. Clarence	1726 Eye St., N.W.
Selinger, Maurice A.	1726 Eye St., N.W.
FLORIDA	
Jacksonville	
Dyrenforth, L. Y.	1022 Park St.
Kirk, Wm. W.	208 Laura St.
Royce, Clayton E.	1022 Park St.
Miami	
Youmans, Iva C.	653 S. W. 2d St.
Tampa	
Mills, Herbert R.	706 Franklin St.
GEORGIA	
Atlanta	
Ayers, A. J.	384 Peachtree St.
Bishop, Everett L.	384 Peachtree St.
Klugh, Geo. F.	139 Forrest Ave., N. E.
Norris, Jack C.	50 Armstrong St.

NAME — ADDRESS

Augusta
Pund, Edgar R.....Univ. of Ga. Sch. of Med.

Emory University
Kracke, Roy R.....Emory University Hospital

Macon
Saye, Ernest B.....820 New St.

ILLINOIS

Bloomington
Markowitz, Benj.....219 N. Main St.

Chicago
Arkin, Aaron.....55 E. Washington St.
Benjamin, Eustace L.....185 N. Wabash Ave.
Brown, Seth E.....533 Grant Pl.
Croy, C. Churchill.....25 E. Washington St.
Davidsohn, Israel.....2750 W. 15th Pl.
Delaney, P. Arthur.....8114 Euclid Ave.
Dwyer, Thomas L.....232 W. 63d St.
Fishback, Hamilton R....303 E. Chicago Ave.
Gardner, Stella M.....30 N. Michigan Ave.
Hennemeyer, R. J.....1305 E. 63d St.
Hill, Lewis R.....1120 Leavitt St.
Hirsch, Edwin F.....1439 S. Michigan Ave.
Howell, Katharine M.....29th and Ellis Ave.
Jaffe, Richard H.....533 Grant Pl.
Kearns, Jerry Joseph.....4458 Madison St.
Kirschbaum, Jack D...Cook County Hospital
Kremer, Rudolph J.....333 Belden Ave.
Levine, Victor.....6104 Woodlawn Ave.
Levinson, Samuel A.....1817 W. Polk St.
Lewis, Julian H.....4750 Champlain St.
Lincoln, Mary C.....30 N. Michigan Ave.
Matthies, M. M.....33 N. Wabash Ave.
Moore, Josiah J.....55 E. Washington St.
Murphy, Leonard J.....4753 Broadway
Neiman, Benj. H.....Cook County Hospital
Nicoll, Homer K.....122 S. Michigan Ave.
Petersen, A. S. J.....45-67 W. 111th St.
Petersen, Wm. F.....1817 W. Polk St.
Pilot, Isadore.....185 N. Wabash Ave.
Pribram, Ernest.....4458 Malden St.
Rosenthal, Sol Roy.....1853 W. Polk St.
Rukstinat, George J.....1758 W. Harrison St.
Saphir, Otto.....29th and Ellis Ave.
Simonds, James P.....303 E. Chicago Ave.
Swan, Mary H.....55 E. Washington St.
Sweany, Henry C.....5601 N. Crawford Ave.
Thalhimer, Wm.....29th and Ellis Ave.
Weiss, Emil.....Univ. of Ill., Coll. of Med.
Wells, H. Gideon.....
.....Dept. of Path., Univ. of Chicago

Decatur
Melnick, Perry J.....
.....Decatur and Macon County Hospital

Evanston
Gunn, Francis D.....2650 Ridge Ave.
Schultz, Oscar T.....355 Ridge Ave.

Joliet
Wilson, W. Henry.....204 Scott St.

Moline
Vollmer, Maud J.....1630 Fifth Ave.

Oak Park
Piette, Eugene C.....518 N. Austin Blvd.

Peoria
Bohrod, Milton G.....124 Randolph St.

Quincy
Cohen, Frank.....529 Hampshire St.

Rockford
Palmer, Harold D.....507 Chestnut St.

Springfield
Bain, Walter G.....8th and Mason Sts.

INDIANA

Bluffton
Nickel, Allen A. C.....Caylor-Nickel Clinic

Evansville
McGlumphy, Chas. B.....614 Mary St.
Seitz, Chas. L.....412 S. E. 4th St.

Fort Wayne
Rhamy, Bonnelle W..... 347 W. Berry St.

Indianapolis
Banks, Horace M.....1604 N. Capitol Ave.
Forry, Frank...Indiana Univ. Medical Center
Langdon, Harry K.....23 E. Ohio St.
Long, Alfred G.....902 E. 49th St.
Thornton, Harold C.Indianapolis City Hospital

Lafayette
Hunter, Frank P.....300 Main St.

Muncie
Cole, Russell E.....203 Western Reserve Bldg.

South Bend
Giordano, Alfred S.....531 N. Main St.
Lyon, Marcus W., Jr...122 N. Lafayette Blvd.

Terre Haute
Selsam, Etta Barkdoll.....221 S. 6th St.

IOWA

Cedar Rapids
Mulsow, Fredk. W.....120, 3d St., S. E.

Cherokee
Pope, John M.....

Clinton
Boyer, Edward E. H.....114, 32d Ave., N.

Davenport
Lamb, Frederick H.....220 Main St.

Des Moines
Weingart, Julius S.....406, 6th Ave.

Dubuque
McNamara, Frank P.....1596 Delhi St.

Iowa City
Herrmann, Walter W.....University of Iowa
Smith, Harry P..132 Medical Laboratory Bldg.
Warner, Emory D.....
.....124 Medical Laboratory Bldg.

Ottumwa
Hecker, Friedrich A.....130 E. Maple Ave.

Sioux City
Starry, Allen C.....St. Joseph's Mercy Hosp.

KANSAS

Kansas City
Wahl, Harry R.....Bell Memorial Hosp.
Wells, Arthur H.....Bethany Methodist Hosp.

Salina
Moses, Howard N.....100 S. Santa Fe St.

Topeka
Lattimore, John L.....901 Kansas Ave.

Wichita
Hellwig, C. Alexander....928 N. Emporia Ave.

KENTUCKY

Lexington
Maxwell, Elmer S.....190 N. Upper St.

Louisville
Allen, John D.....608 S. 4th St.
Gordon, Harold.....Univ. of Louisville
McNeill, Clyde.....321 W. Broadway
Miller, Aura J.....323 E. Chestnut St.
Weeter, Harry M.....332 W. Broadway

Pewee Valley
Peters, John R...Pewee Valley Sanit. & Hosp.

LOUISIANA

Baton Rouge
Beven, John L.....
.....Our Lady of the Lake Sanitarium

Lake Charles
Hebert, Louis A.....834 Ryan St.

Monroe
Pracher, John.....301 Jackson St.

New Orleans
Bowden, Margaret P. H.....210 Baronne St.
Couret, Maurice J.....2000 Tulane Ave.
Friedrichs, Andrew V.....921 Canal St.
Hauser, George H.....927 Canal St.
Hosoi, Kiyoshi.....Tulane Univ. Sch. of Med.
Johns, Foster M.....927 Canal St.
Lanford, John A.....3500 Prytania St.
Lawson, Edwin H.....2700 Napoleon Ave.
Maher, Aldea.....228 St. Charles St.
Ogden, M. A.....2226 Ursuline Ave.

Shreveport
Butler, Willis P.....941 Margaret St.
Ellis, Fredk. G.....624 Travis St.

MAINE

Bangor
Thompson, H. E.....250 State St.

Lewiston
Beliveau, Romeo A..St. Mary's General Hosp.
Gottlieb, Julius.....49 Central Ave.

Portland
Warren, Mortimer.....22 Arsenal St.

MARYLAND

Baltimore
Acton, Conrad.....101 W. Read St.
Collenberg, Henry T.....2 W. Read St.
Gichner, Manuel G.....2426 Eutaw Pl.
Maldeis, Howard J.....104 W. Madison St.
Spencer, Hugh R.....University of Maryland

Hampstead
Bringman, Gladys H.....

MASSACHUSETTS

Boston
Belding, David L.....80 E. Concord St.
Branch, Chas. F.....80 E. Concord St.
Burnett, Francis L.....205 Beacon St.
Flashman, David H.....37 Schuyler St.
Hinton, Wm. A.....25 Bennet St.
Hooker, Sanford B.....80 E. Concord St.
Leary, Olga Cushing.....43 Bay State Rd.
Leary, Timothy.....43 Bay State Rd.
Mallory, Tracy B.....
.....Massachusetts General Hospital
Oslin, J. Edwin.....30 Huntington Ave.
Rooney, James Stewart....53 Parker Hill Ave.
Schlesinger, Monroe J....330 Brookline Ave.
Steele, Albert E.....23 Bay State Rd.
Ulrich, Helmuth.....30 Huntington Ave.
Warren, Shields.....195 Pilgrim Rd.

Bradford
Bartlett, Bernice A.....11 Haseltine St.

Brookline
Dalrymple, S. C.....233 Walnut St.

Fall River
Peasley, Elmus D.....1820 Highland Ave
Walsh, James H.....538 Prospect St.

Lowell
Rodger, James Y.....226 Central St.

New Bedford
Wason, Isabel Mary.....14 Rotch St.

Pittsfield
Criscitiello, Modestino.....8 Bank Row
Scoville, Helen M.....741 North St.

Springfield
Dwyer, John E.....146 Chestnut St.
Jones, Fred D.....20 Maple St.

Westboro
Pierce, Lydia Baker....Westboro State Hosp.

Worcester
Elliott, William J.....119 Belmont St.
Freeman, William.....P.O. Box 489
Goodale, Raymond H....Worcester City Hosp.
Looney, Joseph M....Worcester State Hospital

MICHIGAN

Ann Arbor
Bugher, John C..Dept. of Path., Univ. of Mich.
Howard, S. C.....326 N. Ingalls St.
Wanstrom, Ruth C.....
.....Dept. of Path., Univ. of Mich.
Weller, C. V...Dept. of Path., Univ. of Mich.

Battle Creek
Humphrey, Arthur A.....
.....Leila Y. Post Montgomery Hospital
Lewis, Welcome B....Battle Creek Sanitarium
Roth, Paul.....Battle Creek Sanitarium

Bay City
Gamble, Wm. G., Jr.....2010, 5th Ave.

Detroit
Amolsch, Arthur L..Wayne Univ., Coll. of Med.
Beaver, Donald C.....Woman's Hospital
Brines, Osborne A.....Receiving Hospital
Clark, Harry L.....5057 Woodward Ave.
Cope, Henry E.....1551 Woodward Ave.
Davis, James E.....1512 St. Antoine St.
Hartman, Frank W.....Henry Ford Hosp.
Kasper, Jos. A.....1151 Taylor Ave.
King, Walter E.....Research & Biological Laboratory, Parke, Davis & Co.
Morse, Plinn F.....Harper Hospital
Owen, Clarence I.....4160 John R St.
Owen, R. G.....1551 Woodward Ave.
Stafford, Frank W.....1111 Griswold St.

Eloise
Gould, Sylvester E.....
.....Eloise Hospital and Infirmary

Flint
Backus, Glenn R.....901 Begole St.

Grand Rapids
Bond, Geo. L.....114 Fulton St., E.
German, Wm. M...Blodgett Memorial Hospital

Grosse Pointe
Gruhzit, Oswald M.....580 Hampton Rd.

Kalamazoo
Prentice, Hazel R.....3404 Oakland Dr.

Pontiac
Olsen, Richard E.....900 Woodward Ave.

Saginaw
Lohr, Oliver W.....302 S. Jefferson St.

MINNESOTA

Duluth
Berdez, George Louis.....St. Mary's Hospital

Minneapolis
Baker, Looe.....1111 Nicollet Ave.
Drake, Charles R.....900 Nicollet Ave
Grave, Floyd.....823 Nicollet Ave.
Lufkin, Nathaniel H.....
.....424 W. Diamond Lake Rd.
Merkert, Geo. L.....1412 E. 24th St.
Smith, Margaret I.....2627 Chicago Ave.

Rochester
Kernohan, J. W.....Mayo Clinic
MacCarty, Wm. C.....Mayo Clinic
Magath, Thos. B.....Mayo Clinic
Robertson, H. E.....Mayo Clinic
Rosenow, E. C.....Mayo Clinic
Sanford, Arthur H.....Mayo Clinic
Wellbrock, Wm. L. A.....Mayo Clinic
Wilson, Louis B.....Mayo Foundation

St. Cloud
Stangl, Fred H.....101 7th Ave., S.

NAME — ADDRESS

St. Paul
Ikeda, Kano.....125 W. College Ave.
Noble, John Franklin.....Ancker Hospital

MISSISSIPPI

Greenville
White, E. T.....301½ Washington Ave.

Meridian
Krauss, William.....818, 22d Ave.

Vicksburg
Lippincott, Leon S.....920 Crawford St.

MISSOURI

Columbia
Neal, M. Pinson..Dept. of Path., Univ of Mo.

Jefferson City
Adams, Charles F...Mo. State Board of Health

Kansas City
Duncan, Ralph Emerson.....306 E. 12th St.
Hall, Frank J.....306 E. 12th St.
Helwig, Ferdinand C.....St. Luke's Hospital
Johnson, Emsley T.....St. Joseph Hospital
Koritschoner, Robt.....4949 Rockhill Rd.
Narr, Frederick C.....Research Hospital
Stewart, Edward L.....1115 Grand Ave.
Trimble, Wm. K.....1103 Grand Ave.

St. Louis
Allen, Hollis N.....634 N. Grand Blvd.
Buhman, Rudolph.....539 N. Grand Blvd.
Gradwohl, R. B. H.....3514 Lucas Ave.
Harris, D. L.....508 N. Grand Blvd.
Ives, Geo.....3720 Washington Blvd.
Katz, Samuel D.....3720 Washington Blvd.
Klenk, Chas. L.....508 N. Grand Blvd.
McCordock, Howard A.....Washington Univ.
Schery, Chas. Wm.....529 N. Whittier St.
Sickert, Walter J.....Washington Univ.
Thompson, Ralph L.....607 N. Grand Blvd.
Walsh, L. S. Newman.....5535 Delmar Blvd.

Springfield
Stone, Murray C.....200 E. Pershing St.

MONTANA

Butte
Peterson, Raymond F.....57 W. Quartz St.

Great Falls
Hitchcock, E. D.....Great Falls Clinic
Walker, Thos. F.....503, 1st Ave., N.

Poplar
Dahlstrom, Arthur W.....U. S. Indian Hosp.

NEBRASKA

Omaha
Eggers, Harold E.Univ. of Nebr., Coll. of Med.
Manning, E. T.....107 S. 17th St.
Moody, W. B.....105 S. 17th St.
Rubnitz, A. S.....107 S. 17th St.
Russum, B. Carl.....306 N. 14th St.
Tollman, James P.....42nd and Dewey Ave.

NEVADA

Reno
Parsons, Lawrence.....235 W. 6th St.

NEW HAMPSHIRE

Hanover
Miller, Ralph E.....9 Downing Rd.

NEW JERSEY

Arlington
Gilman, C. M. B.....59 Seeley Ave.

Asbury Park
de Pons, Isabel S. C.....501 Grand Ave.
Pons, C. A.....501 Grand Ave.

Atlantic City
Kilduffe, Robt. A.....Atlantic City Hosp.

East Orange
Minier, Carl L.....157 Harrison St.

Elizabeth
Casilli, A. R.....618 Newark Ave.

Englewood
Halpern, Herman.....143 Engle St.

Greystone Park
Christian, Thos. B.....

Jersey City
Alter, Nicholas M.....410 Fairmount Ave.
Hemsath, Fredk. A.....176 Palisade Ave.

Mount Holly
Viteri, Luis E.....137 High St.

Newark
Antopol, Wm.....Beth Israel Hosp.
Brown, Lewis W.....160 Roseville Ave.
Eckikson, Joseph I.....845 S. 12th St.
Goldberg, Samuel A.....27 S. 9th St.
Gray, John W.....142 Clinton Ave
Yaguda, Asher.....88 Clinton Ave.

Orange
Cline, Benj. F.....264 Central Ave.

Paterson
Davis, A. Hobson.....Paterson General Hosp.
Kim, Gay B.....703 Main St.

Plainfield
Borow, Louis S.....934 Park Ave.

Teaneck
Markley, Luther A.....Holy Name Hospital

Toms River
Halbach, Robert McC.....802 Main St.

Trenton
Boughton, Thos. H.....446 Bellevue Ave.
Rogers, Wm. N.....1255 Brunswick Ave.

NEW MEXICO

Albuquerque
Beam, M. P.....221 W. Central Ave.

NEW YORK

Albany
Clemmer, John J.....136 S. Lake Ave.
Gilbert, Ruth.....116 N. Allen St.
Horner, Henrietta Calhoun..171 S. Main Ave.
Klinck, Gustavus H., Jr.....Albany Med. Coll.
Wright, A. W.....Albany Med. Coll.

Binghamton
Bergstrom, V. W.....Binghamton City Hosp.
Gregory, Hugh S.....Binghamton State Hosp.

Brooklyn
Baker, Margaret M.....437 Ovington Ave.
Black, F. A.....80 Hanson Pl.
Derby, Irving Marsh.....681 Clarkson Ave.
Fein, M. J.....50 Greene Ave.
Fink, Harold.....1304 Beverly Rd.
Fradkin, William Z.....955 Eastern Pkwy.
Grainick, Abraham.....119 Sumner Ave.
Greeley, Horace.....140 Clinton St.
Halpert, Béla.....555 Prospect Pl.
Kantrowitz, Abraham R.....475 Ocean Ave.
Lederer, Max.....555 Prospect Pl.
Marten, M. Edward.....515 Ocean Ave.
Moitrier, Wm., Jr.....1219 Dean St.
Morrison, Maurice.....250 Ocean Pkwy.
Nidish, Edward H.....1272 Bergen St.
Polayes, Silik H.....425 Prospect Pl.
Wiener, Alexander S.....520 Crown St.

Buffalo
Bentz, Charles A....126 W. Humboldt Pkwy.
Hanan, Ernest B.....462 Grider St.
Jacobs, William F.....408 Richmond Ave.
Rosedale, Raymond S.....276 Norwalk Ave.
Vaughan, Stuart L.....100 High St.
Warwick, Margaret.....875 Lafayette Ave.
Williams, Herbert U.....30 Arlington Pl.

Central Islip
Trygstad, Reidar.....Central Islip State Hosp.

Clifton Springs
Thomas, Walter S.....42 Kendall St.

Corning
Shafer, Rudolph J.....163 E. 1st St.

Cortland
Wall, Wm. A.....134 Homer Ave.

Elmira
Bleyer, Leo F.....555 E. Market St.
Munch, Otto Louis.....
.....Arnot-Ogden Memorial Hospital
Stuart, Anna M.....656 Park Pl.

Far Rockaway
Handelman-Kalashnikoff, Pauline.....
.....536 Beach 22d St.

Glens Falls
Maslon, Morris.....191 Glen St.

Ithaca
Hauenstein, B. F.....
.....Tompkins Co. Memorial Hospital

Jackson Heights
Angrist, Alfred.....37-35, 74th St.

Jamaica
Buxbaum, Edward J.....8711, 150th St.
Campbell, N. H. M.....89-18, 139th St.
Werne, Jacob.....89-04, 148th St.

Kings Park
Priestman, Gordon.....

Kingston
Taylor, James Spottiswood.....
.....City of Kingston Laboratory

Little Neck
Van Nostrand, Hobart S.....
..... 45-06 Little Neck Pkwy.

Long Island City
Hala, Wm. W.....30-20, 29th St.

Marcy
Bower, George C.....Marcy State Hospital

Middletown
Kelly, Wm. E.....
.....Middletown State Homeopathic Hosp.

Newark
Baumgartner, E. A.....Newark State School

Newburgh
Wescott, A. M.....231 Liberty St.

New Rochelle
Brooks, Henry T.....35 Woodland Ave.
McIlroy, P. T.....421 Huguenot St.

New York
Aronoff, Rosa.....Metropolitan Hospital
Aronson, Wm.....150 E. 182d St.
Block, Nathan.....2280 Andrews Ave.
Brown, Chester R.....150 W. 87th St.
Cocheu, Lindsley F.....205 E. 69th St.
Curphey, Theodore J.....115 E. 61st St.
Darlington, Charles G.....209 E. 23d St.
Dolgopol, Vera B.....131 W. 110th St.
Donnet, J. Victor.....57 W. 57th St.
DuBois, Phebe L.....150 E. 73d St.
Eggston, Andrew A.....653 Park Ave.
Ehrlich, Joseph C.....Lebanon Hospital
Elser, Wm. J.....525 E. 68th St.
Ewing, James.....2 W. 106th St.
Felsen, Joseph.....667 Madison Ave.
Ferraro, Louis R.....711 E. 230th St.
Foot, Nathan Chandler.....525 E. 68th St.
Fraser, Alexander.....153 W. 11th St.
Frosch, Herman L.....1882 Grand Concourse
Geiger, Jacob.....25 Central Park West
Gerber, Isadore E.....1 E. 100th St.
Gonzales, Thomas A.....56 E. 87th St.
Graef, Irving.....477 First Ave.
Grauer, Frank.....226 W. 71st St.
Hadjopoulos, L. G.....6 E. 78th St.
Heitzmann, Louis.....38 W. 90th St.
Helpern, Milton.....975 Park Ave.
Hillman, Oliver S.....140 E. 54th St.
Hochman, Charles H...2715 Grand Concourse
Jacobson, Sheldon A.....1919 Madison Ave.
Jaffe, Henry L.....1919 Madison Ave.
Jeffries, Ferdinand M.....18 E. 41st St.
Jessup, D. S. D.....411 W. 114th St.
Kaliski, David J.....70 E. 83d St.
Klemperer, Margit Freund.....
.....385 Central Park West
Klemperer, Paul.....385 Central Park West
Knox, Lelia Charlton.....St. Luke's Hospital
Kopel, Moses.....1454 Grand Concourse
Larimore, L. D.....750 Riverside Dr.
Lefkowitz, Louis L.....1051 Tiffany St.
Levine, Jacob.....1345 Shakespeare Ave.
Lisa, James R.....New York City Hospital
MacNeal, W. J.....303 E. 20th St.
Manheims, Perry J.....27 W. 96th St.
McNeil, Archibald.....18 E. 41st St.
Meeker, Louise H.....303 E. 20th St.
Moolten, Sylvan E.....60 E. 96th St.
Olcott, Charles T.....1300 York Ave.
Pincus, Julius.....250 W. 75th St.
Price, Aaron S.....335 W. 50th St.
Rabson, S. Milton.....166 W. 72d St.
Rohdenburg, G. L.....111 E. 76th St.
Rosenthal, Nathan.....51 E. 90th St.
Rous, Peyton.....
..Rockefeller Institute for Medical Research
Rubenstein, Morris.....1475 Walton Ave.
Saccone, Andrea.....334 E. 116th St.
St. George, Armin V.....400 E. 29th St.
Seecof, David P.....1970 Daly Ave.
Shuster, Mitchell.....30 E. 40th St.
Smith, Lawrence W..Willard Parker Hospital
Sondern, Frederic E.....20 W. 53th St.
Sophian, L. H.....428 W. 59th St.
Stanford, Addie D.....450 E. 64th St.
Stillman, Ralph G.....525 E. 68th St.
Taub, Jacob.....1574 Leland Ave.
Taylor, Ewing.....27 W. 44th St.
Thro, William C.....1300 York Ave.
Wallerstein, Harry.....585 West End Ave.
Weintraub, Solomon.....240 E. 79th St.
Weiss, M. Arthur.....235 W. 76th St.
Whitcher, Burr R.....305 E. 20th St.

Ogdensburg
Porro, Francis W...A. Barton Hepburn Hosp.

Ossining
Gosline, Harold I.....275 Spring St.

Ozone Park
deVeer, J. Arnold.....101-32, 97th St.

Poughkeepsie
Carpenter, H. P...Hudson River State Hospital
Peckham, A. L.....Vassar Brothers Hosp.

Rochester
Brown, Herbert R.....224 Alexander St.
Gáspár, István A.....501 W. Main St.
Hawkins, William B....260 Crittenden Blvd.
Kennedy, Robert P.....176 S. Goodman St.
Lindsay, Saml. T.....909 W. Main St.
O'Grady, Geo. W.....277 Alexander St.

Rye
Loder, M. M.....

Saranac Lake
Gardner, L. U.....7 Church St.

Schenectady
Kellert, Ellis.....Ellis Hospital

Syracuse
Ferguson, J. Howard.....309 S. McBride St.
Weiskotten, H. G.....309 S. McBride St.

NAME — ADDRESS

Troy
Curry, A. Hazel.....467 Pawling Ave.
Curtis, Stephen H.....Leonard Hospital
Jacobsen, V. C.....51, 1st St.

Utica
Gallagher, C. D.....1676 Sunset Ave.
Russell, Clarence L.....1125 Court St.

Valhalla
Dalldorf, Gilbert J.....Grasslands Hospital
Springer, Joyce M.....Grasslands Hospital

Watertown
Walker, Thomas T.....11 Public Sq.

Westfield
Field, Cyrus W.....88 N. Portage St.

White Plains
Russell, Hollis K.....82 Concord Ave.

Yonkers
Cook, Ward H.....Bureau of Laboratories
Weedon, Frederick R..Bureau of Laboratories

NORTH CAROLINA

Asheville
Crump, Curtis..... 36 Grove St.

Chapel Hill
Bullitt, James B.....

Charlotte
Barret, Harvey P.....403 N. Tryon St.
Todd, Lester C.....403 N. Tryon St.

Durham
Baker, Roger D.....Duke Hospital
Byrnes, Thomas H.....Watts Hospital
Forbus, Wiley D.....Duke Hospital
Sprunt, Douglas H.....Duke Hospital

Wake Forest
Carpenter, C. C...Wake Forest School of Med.

Wilmington
Barefoot, Graham B.....404 N. 3d St.

NORTH DAKOTA

Bismarck
Larson, L. W.....221, 5th St.

Grand Forks
Saiki, A. K.....Univ. of N. Dak. Med. Sch.

Minot
Breslich, Paul J.....Trinity Hospital

OHIO

Akron
Hathaway, Burr M.....Children's Hospital
Potter, Frederick C.....256 W. Cedar St.
Saylor, Edward L.....525 E. Market St.

Cincinnati
Faller, Albert.....19 W. 7th St.
Herzberg, Mortimer.....Jewish Hospital
Patterson, James N.....Univ. of Cincinnati
Zeek, Pearl M.....Cincinnati Gen. Hosp.

Cleveland
Dominguez, Rafael.....2272 S. Taylor Rd.
Goldblatt, Harry.....2085 Adelbert Rd.
Karsner, Howard T.....2085 Adelbert Rd.
Kline, Benjamin S.....1800 E. 105th St.
Moritz, Alan R.....2085 Adelbert Rd.

Columbus
Coons, J. J.....370 E. Town St.
Fidler, Roswell S.....700 N. Park St.
Hoffman, Ralph W.....1542 W. 1st Ave.
Reinhart, Harry L.....1711 Essex Rd.
Shilling, Ellis Ray.....345 E. State St.

Dayton
Payne, Foy C.....201 S. Main St.
Simpson, Walter M.....134 Apple St.

Dover
Shaweker, Max.....Reeves Bank Bldg.

Elyria
Rosenzweig, Maurice.....630 E. River St.

Lorain
Donaldson, John B.....700 Broadway

Newark
Mitchell, Louis A.....28 E. Locust St.

Springfield
Oesterlin, Ernest J.....Springfield City Hosp.

Toledo
Crumrine, Ralph M.....
.....Lucas County General Hospital
Hindman, S. S.....316 Michigan St.
Ramsey, Thomas L.....225 Michigan St.
Rucker, James B.....630 W. Central Ave.
Schade, August H.....320 Michigan St.
Steinberg, Bernhard.....Toledo Hospital
Zbinden, Theodore.....706 Madison Ave.

Youngstown
Kramer, G. B.....Youngstown Hospital

OKLAHOMA

Bartlesville
Chamberlin, E. M.....
.....Washington County Memorial Hosp.

El Reno
Muzzy, Wm. J.....212 S. Evans St.

Oklahoma City
Bailey, Wm. H.....300 W. 12th St.
Jeter, Hugh G.....1200 N. Walker St.
Keller, W. F.....119 N. Broadway

Tulsa
Nelson, I. A.....108 W. 6th St.
Venable, Sidney C.....420 S. Main St.

OREGON

Eugene
Furrer, Emil D.....130 E. Broadway

Portland
Foskett, H. H.....1058 S. W. Taylor St
Hunter, Warren C..Univ. of Oregon Med. Sch.
Lawrence, H. J.....322 Alder St.
Manlove, Chas. H...2266 Marshall St., N. W.
Menne, Frank R...Univ. of Oregon Med. Sch.
Robertson, Thomas D....1025 N.W. 24th Ave.

PENNSYLVANIA

Abington
Elman, John.....Abington Memorial Hosp.

Allentown
Hodgens, Helen W...Allentown State Hospital
Milstead, L. C.....4th and Chew Sts.
Wenner, John J.....941 Hamilton St.

Altoona
Brumbaugh, A. S.....1312, 11th St.

Ardmore
Belk, William P.....Times Med. Bldg.

Bethlehem
Rothrock, Henry A., Jr....St. Luke's Hospital

Chester
Sickel, Geo. B.....525 Welsh St.

Danville
Hunt, Henry F.....

Easton
Gaines, Carl.....130 N. 3d St.
Zillessen, F. O.....256 Bushkill St.

Erie
Armstrong, E. L.....2d and State Sts.

Fortyfort
Janjigian, R. R.....1043 Wyoming Ave.

Gettysburg
Stewart, Henry.....

Greensburg
Mayhew, J. Morgan.....532 W. Pittsburgh St.

Harrisburg
Denison, Charles M..Harrisburg State Hospital
Moffitt, George R.....Harrisburg Hospital
Van Horn, Herman H.....
.....3d St. and Polyclinic Ave.

Huntingdon
Reiners, Charles R.....741 Washington St.

Johnstown
Anderson, Horace B.....218 Franklin St.
McCloskey, B. J.....1020 Franklin St.

Kingston
Daley, D. F.....214 Chestnut St.
Wenner, Thos. J.....562 Wyoming Ave.

Lancaster
Keasbey, Louisa E.....Lancaster Gen. Hosp.

Lansdowne
Kennedy, Patrick J.....65 Fairview Ave.

Mayview
Fetterman, George H.....
.....Pittsburgh City Home and Hospitals

McKeesport
Menlowe, P. M.....1231 Evans Ave.
Sandblad, A. G.....1701 Union St.

New Brighton
McLaren, Harold J.....541, 11th Ave.

Norristown
Laubach, Charles A..Norristown State Hospital
Simpson, John C.....920 Swede St.

Philadelphia
Asnis, Eugene J.....1524 Chestnut St.
Barthmaier, O. F.....2303 W. Lehigh Ave.
Bauer, John T.....8th and Spruce Sts.
Becker, Carl Emil.....2341 N. College Ave.
Brown, Claude P. .....1930 Chestnut St.
Bucher, Carl Joseph.....271 S. 15th St.
Case, Eugene Allen.....1818 Lombard St.
Clark, J. H.....3701 N. Broad St.
Corson-White, E. P.....1820 Pine St.
Crawford, Baxter L.....
.....Jefferson Med. Coll. and Hosp.
Custer, Richard P.....4035 Chestnut St.
de Rivas, Damaso.....1831 Chestnut St.
Fisher, H. Russell.....226 W. Gorgas Lane
Fowler, Kenneth.....Presbyterian Hospital
Fox, Herbert..Pepper Laboratory, Univ of Pa.
Gault, Edwin S.....Temple Univ. Sch. of Med.
Ginsburg, Gershon.....1936 N. 7th St.
Hastings, Willard S.....Jeanes Hosp.
Hopp, George A.....20th and Chestnut Sts.
Konzelmann, F. W....Broad and Ontario Sts.
Krumbhaar, E. B...Univ. of Pa. Sch. of Med.
Long, Esmond R.....Henry Phipps Institute
Lucké, Balduin....Univ. of Pa. Sch. of Med.
Lynch, Frank B., Jr.....
.....Germantown Disp. and Hosp.
McFarland, Joseph.....
.....McManes Laboratory, Univ. of Pa.
Meranze, David R.....Mt. Sinai Hosp.
Moon, V. H..Jefferson Med. Coll. and Hospital
Rees, William T.....3763 N. 18th St.
Reimann, S. P.....Lankenau Hospital
Richardson, Russell.....320 S. 16th St.
Rose, S. Brandt.....4035 Chestnut St.
St. John, E. Quintard.....1833 Chestnut St.
Soloff, Louis A.....St. Joseph's Hospital
Spaeth, William L. C.....5000 Jackson St.
Steinberg, Wm.....1818 Pine St.
Stewart, Harold L.....
.....Jefferson Med. Coll. and Hospital
Tuft, Louis.....1530 Locust St.
Zeckwer, Isolde Therese.....
.....Univ. of Pa. Sch. of Med.

Pittsburgh
Baker, Moses H.....121 University Pl.
Bruecken, A. J.....St. Francis Hospital
Cohen, Mortimer .....
.....Univ. of Pittsburgh Med. Sch.
Grauer, Robert C.....2001 Murray Ave.
Gross, Paul.....4800 Friendship Ave.
Hamilton, Robt. C.....Passavant Hospital
Haythorn, Samuel R..210 E. Park Way, N. S.
Joyce, Francis W.....4001 California Ave.
Lacy, George Rufus.....
.....Univ. of Pittsburgh Med. Sch.
McClellan, Robt. H.....265, 46th St.
McMeans, J. W.....Pittsburgh Hospital
Mellon, Ralph R.....6055 Bunkerhill Dr.
Moyer, Ray P.....1021 Portland St.
Permar, Howard H.....Mercy Hospital
Ray, Henry M.....500 Penn Ave.
Rockman, Jacob.....2117 Carson St.
Ross, Elizabeth.....5230 Centre Ave.
Wallhauser, Andrew.....Univ. of Pittsburgh
Willetts, Ernest W.....429 Penn Ave.
Yardumian, K.....Montefiore Hospital

Pottsville
Moll, Francis K.....309 Mahantongo St.

Reading
Elton, Norman W.....2340 Perkiomen Ave.
Funk, Erwin Deaterly.....Reading Hospital

Sayre
DeWan, Chas. H.....Robert Packer Hosp.

Scranton
Clark, Geo. A.....129 Washington Ave.
Cooper, Harold B.....633 E. Market St.

Sewickley
Feltwell, Myrtle R.....

Sharon
Hartman, Geo. O.....740 E. State St.

Torrance
Wiseman, John Ignatius.....

Warren
Eaton, H. C.....Warren State Hospital

Washington
Ramsey, G. W.....Washington Hospital

West Chester
Hollingsworth, I. P. P....411 N. Walnut St.

Wilkes-Barre
Jamieson, Howard M.....
.....Wilkes-Barre General Hospital

York
Pusch, Lewis C.....York Hospital

RHODE ISLAND

Howard
Crank, Rawser P.....
.....State Hospital for Mental Diseases

Providence
Clarke, B. Earl.....593 Eddy St.
Hamilton, James.....349 Hope St.
Schradieck, C. E.....825 Chalkstone Ave.

SOUTH CAROLINA

Charleston
Johnson, Francis B.....16 Lucas St.
Lynch, Kenneth M.....16 Lucas St.

Columbia
Plowden, Henry H.....2020 Hampton St.
Smith, Herbert M.....State Office Bldg.

Greenville
Wilson, Thos. R. W.....109 Memminger St.

Spartanburg
Mosteller, Ralph.....855 N. Church St.

SOUTH DAKOTA

Vermillion
Ohlmacher, Joseph C.....309 Lewis St.

NAME — ADDRESS

TENNESSEE

Chattanooga
Crowell, Tolbert C.....544 McCallie Ave.

Knoxville
DePue, Ray V.....601 Walnut St.
Monger, Ralph H.....603 W. Main Ave.

Memphis
Jones, Maurice L.....
.....Pathological Institute, Univ. of Tenn.
Leake, Nolan E.....399 Madison Ave.
McIntosh, J. A. Jr.....264 Jackson Ave.
Moss, Thomas C.....1265 Union Ave.
Schmittou, L. V.....130 Madison Ave.

Nashville
Jones, Robt. L.....706 Church St.
Rigdon, Raymond H.....Vanderbilt Hosp.

TEXAS

Austin
Bohls, Sidney W .....410 E. 5th St.
Graham, G. M.....110 W. 7th St.
Jackson, J. Warren.....110 W. 7th St.

Beaumont
Lewis, Seaborn J.....398 Pearl St.
Williford, H. B.....595 Orleans St.

Dallas
Bell, Marvin D.....1719 Pacific Ave.
Black J. Harvey.....1719 Pacific Ave.
Brandes, W. W....Baylor University Hospital
Caldwell, Geo. T....Baylor Univ. Coll. of Med.
Carter, Chas. F.....1719 Pacific Ave.
Goforth, John L.....3121 Bryan St.
Hill, Joseph M.....Baylor University Hospital
Sanders, C. B....Oak Lawn and Maple Aves.
Wallace, Stuart A.....3301 Junius St.

El Paso
Dutton, Loraine Orr.....114 Mills St.
Turner, Geo.....109 N. Oregon St.
Waite, Willis W.....114 Mills St.

Fort Worth
Hulsey, Sim.....600 W. 10th St.
Owen, May.....600 W. 10th St.
Terrell, Truman C.....600 W. 10th St.

Galveston
Brindley, Paul.....900 Avenue B
Marr, William L.....900 Avenue B

Houston
Braden, Albert H.....1910 Crawford St.
Braun, Harry E.....Jefferson Davis Hospital
Wood, Martha A.....1215 Walker Ave.

Jacksonville
Sory, Wm. H....Nan Travis Memorial Hospital

San Antonio
Moore, John M.....705 E. Houston St.
Scott, Raymond E.....705 E. Houston St.
Stout, Beecher F.....705 E. Houston St.

Temple
Phillips, Charles.....1302 N. 5th St.
Robinson, James E.....304 S. 22d St.

Wichita Falls
Glover, Milton H.....900, 8th St.
Venable, Douglas R.....2010 Garfield St.

UTAH

Ogden
Schelm, Geo. W.....
.....Thomas D. Dee Memorial Hospital

Salt Lake City
Flood, Thos. A.....8 E. 3d South St.
Ogilvie, O. A.....50 E. South Temple St.

VERMONT

Burlington
Buttles, Ernest H.....Mary Fletcher Hospital

VIRGINIA

Norfolk
Motyca, Lawrence J.....
.....Norfolk Protestant Hospital
Roche, Mary E.....229 W. Bute St.

Richmond
Beck, Regena C.....1103 W. Franklin St.
Broders, A. C.....Medical College of Va.
Budd, Saml. W.....1000 W. Grace St.
Shaw, Frederick W.....Medical College of Va.

WASHINGTON

Seattle
Cefalu, Victor.....509 Olive St.
Magnusson, G. A.....509 Olive St.
Nickson, D. H.....803 Summit Ave.

Sedro Woolley
West, P. C.....Northern State Hospital

Spokane
Edgar, James D.....733 W. 4th Ave.
Patton, Frank R.....407 Riverside Ave.
Patton, Matthew M.....407 Riverside Ave.
Stier, Robt. F. E.....407 Riverside Ave.

Tacoma
McColl, Charles R.....1812 S. Eye St.
Terry, Benjamin T...Tacoma General Hospital

WEST VIRGINIA

Bluefield
Grant, Margaret S.....1710 Bland St.
Sinclair, M. W.....204 Ramsey St.

Charleston
Beck, James S. P..Charleston General Hospital
Gillies, McLeod.....812 Beech Ave.

Clarksburg
Light, Frederick W.....St. Mary's Hospital

Huntington
Hodges, Frank C.....955, 4th Ave.

Morgantown
Fenton, C. C.....West Virginia Univ.

Wheeling
Little, Harold G.....2000 Eoff St.

WISCONSIN

Eau Claire
Scullard, Garner.....219 Roosevelt Ave.

LaCrosse
Thurston, Eric W.....St. Francis Hospital

Madison
Bayley, William E.....1300 University Ave.
Bunting, C. H.....426 N. Charter St.
McGary, Lester.....925 Mound St.
Pessin, S. B.....720 S. Brooks St.
Ritchie, Gorton.....426 N. Charter St.
Stovall, Wm. D....Service Memorial Institute

Milwaukee
Barta, Edward F.....2766 N. 40th St.
Enzer, Norbert.....425 E. Wisconsin Ave.
Fernan-Nunez, Marcos.....561 N. 15th St.
Grill, John.....561 N. 15th St.
Hansmann, G. H.....3321 N. Maryland Ave.
Seelman, John J.....205 E. Wisconsin Ave.
Tharinger, E. L.....231 W. Wisconsin Ave.

Oshkosh
Dickelmann, Lorin E.....46 Washington Blvd.

CANAL ZONE

Ancon
Bates, Lewis B.....Gorgas Hospital

HAWAII

Honolulu
Fennel, Eric A.....
.....The Clinic, Young St. at Thomas Sq.
Larsen, Nils P.....Queen's Hospital

Koloa
Ecklund, Archibald M.....

PHILIPPINE ISLANDS

Manila
de Leon, Walfrido.....609 Kansas Ave.

PUERTO RICO

San Juan
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SATURDAY, OCTOBER 26, 1935

RESEARCH ON THE ELEMENTARY BODIES IN VIRUS DISEASE

Among the serious diseases that affect man and still engage the curiosity of medical research are those believed to be caused by viruses. Some of these have prevailed as world wide epidemics. Others through frequent localized outbreaks have taken the lives of countless children and left others permanently injured. Our knowledge of methods of prevention of some of the virus diseases has advanced more rapidly than the knowledge of their causes.

In 1904, Borrell observed enormous numbers of minute bodies in scrapings from the lesions of fowlpox. Two years later, Paschen observed similar minute bodies in material from vaccinia and smallpox. Little attention was given to these observations for about twenty-five years for the simple reason that these minute bodies were thought likely to be artefacts or protein aggregates. Recently, however, intense research has been focused on the minute elementary bodies in virus diseases by numerous investigators. Like others, Ledingham [1] of the Lister Institute of London entered this field with prejudice against the importance of the elementary bodies. Finally he became satisfied that such bodies were present in suitably stained material, and he followed their variations in number and location as the virus disease progressed. In 1931 he reported that the elementary bodies of Paschen were demonstrable in large numbers on the second or third day in material from the rabbit lesion, while on subsequent days they decreased in number, with a tendency to congregate in the neighborhood of cells of the lesion, especially the polymorphonuclear cells. The next objective was to obtain these bodies from the raw material in pure suspension. With the use of ether and the centrifuge it was possible to separate sharply the epithelial and the degenerated polymorphonuclear cells from the other constituents of the raw material, leaving an opalescent fluid below. When centrifugated again this fluid yielded a deposit extremely rich in elementary bodies. By continuing the process of fractional centrifugation, he obtained a fairly pure suspension of Paschen elementary bodies. Then it was shown that an animal injected with a single dose of virus intradermally developed, about the fifth or sixth day, agglutinins in the blood serum, which rose to a maximum and then slowly declined, exactly as agglutinins do in response to a dose of bacteria.

When pure suspensions of elementary bodies are wanted for infectivity tests on animals, a somewhat different technic must be followed to avoid contact with ether. Instead, the raw virus-containing material from the lesion is roughly centrifugated, then filtered through a Berkefeld V filter, and the filtrate centrifugated at high speed (15,000 revolutions per minute) for at least half an hour. The great mass of elementary bodies will thus be thrown down. With this technic, Ledingham and his associates obtained suspensions of the elementary bodies in vaccinia and carried out infection experiments. They showed that a powerfully infective vaccinia filtrate could be completely depleted of its infective power by means of the high speed centrifuge; furthermore, that the deposit of elementary bodies thrown down by the centrifuge alone proved to be infective. These experiments have been repeated and confirmed by other investigators. The conclusion seems warranted that the elementary bodies are almost surely the virus agents of vaccinia.

This method of experimentation has been applied in research on variola, varicella and herpes zoster, and probably it will be extended to more of the virus diseases. Amies, a colleague of Ledingham, has conducted serologic tests with the elementary bodies. The material from variola minor was inoculated into the skin of the monkey, and a suspension of the elementary bodies from the lesion was prepared; a suspension was prepared also from the dermal reaction in the rabbit in response to variola virus. It was found that the serums of animals experimentally infected with variola minor produced little or no agglutination of the elementary bodies prepared from vaccinia lesions, whereas in a high proportion of the experiments these serums agglutinated the elementary bodies obtained from variola material. Singularly, monkeys infected with variola developed agglutinins for the elementary bodies of variola but not for vaccinia, whereas rabbits inoculated with material from these monkeys developed agglutinins both for variola and for vaccinia. Some common antigen, therefore, is shared by variola and vaccinia. The precise change that takes place in the antigen when variola assumes by passage the attenuated form of vaccinia, however, must be left to future research by means of absorption experiments.

Amies also studied the elementary bodies in varicella. Fluid from the earliest vesicles was placed in a citrated saline solution and cleared by centrifugation at low speed; the supernatant fluid was then centrifu-

1. Ledingham, J. C. G.: Studies on Virus Problems, Bull. Johns Hopkins Hosp. **57**: 32 (July) 1935.

gated at high speed and the elementary bodies thrown down. To get sufficient elementary bodies many vesicles had to be pricked, as only those which gave clear fluid could be used. Since in varicella the agglutinins that appear about five days after the onset of the eruption persist for some time, it may be possible by means of agglutination tests with suspensions of varicella and variola elementary bodies to decide doubtful cases of smallpox when varicella is suspected.

Some intimate connection between varicella and herpes zoster has been indicated for years by clinical and epidemiologic evidence. It is not unusual for varicella to occur among contacts of a case of zoster, and there appear to be authentic records of zoster following exposure to varicella. By the foregoing methods of isolation of elementary bodies and the agglutination test, Amies obtained agglutination of the corresponding elementary bodies in all but two of thirty-two cases of herpes zoster, while in nineteen of them the serums also agglutinated varicella elementary bodies.

Since similar results have been secured by others, there is at last some agreement that many viruses are represented by visible and workable elementary bodies. There lies ahead the goal to add much more knowledge of the behavior of each virus in the host and, if possible, its cultivation in more artificial conditions outside the host. The present available knowledge of viruses is indeed limited. At the moment, for example, where do we stand on the most crucial question, Are these viruses really independent living agents?

TRAUMATIC SHOCK

A large volume of literature has accumulated on the subject of traumatic shock [1] and numerous theories concerning its etiology have been advanced. The obvious picture of prime importance among the phenomena attending shock is the low arterial pressure; two theories for the explanation of this condition have generally received the most attention. One suggests a traumatic toxemia, with the absorption of metabolic toxins from the traumatized areas, followed by a secondary generalized vasodilatation and augmented permeability of the capillaries. The other hypothesis that has been emphasized supports the contention that the condition of shock is caused by the local loss of blood and/or plasma, resulting in a marked decrease in the volume of circulating blood. Hemorrhage is the simplest and most direct method of reducing blood volume, and the similarities between traumatic shock and the results of serious hemorrhage have led observers to remark that the conditions of hemorrhage and shock are identical. This statement is in agreement with the evidence now available. In addition to the factors mentioned in the production of traumatic shock, it seems highly probable that certain nervous influences are of importance. There is the possible inhibition of vasoconstrictors and stimulation of vasodilators in acute circulatory collapse and, what is even more likely, a prolonged activity of the sympathetic system when circumstances arise which bring that system into operation.[2]

Although the evidence supporting the circulatory theory of traumatic shock is in general in good agreement, much confusion exists with respect to efforts designed to support the "toxic theory" of the etiology of traumatic shock. The carefully controlled work of Cannon and Bayliss [3] produced data which suggest that a toxic factor is of importance in shock experimentally induced in cats. However, there is considerable evidence against the theory of traumatic toxemia.[2] The evaluation of the experimental results is made quite difficult because they have been obtained with a variety of animal species and with the use of many diverse methods for the preparation of extracts containing the blood volume reducing toxic factor. In fact, it is possible to produce from a wide variety of normal tissues extracts which contain both pressor and depressor substances.[4] There has been a lack of convincing, positive evidence that a vasodepressive toxin derived from dying tissues induces shock or, on the other hand, conclusive proof that such toxic material could not exist.

Efforts to obtain results of the latter type have recently been reported by Roome and Wilson.[5] A careful study was made of the effects on the blood pressure of extracts from traumatized limbs. As previous experiments of this type frequently have employed tissue extracts obtained by chemical and nonphysiologic methods which might alter materials present in an extract, the University of Chicago investigators have used a rapid and entirely mechanical method for extraction of traumatized muscle. The latter tissue was used, since the cases of large injuries to muscles are those in which shock most commonly occurs. Precautions were taken to purify the extracts by centrifugation, thus removing particles of fat and tissue which might cause sudden death by pulmonary embolism. Furthermore, the animals used for the injections were heparinized to vitiate possible blood-coagulating properties of the extracts. The latter were administered by a viviperfusion method which prevented alterations in the volume of circulating blood. With this experimental technic it was observed that intravenous injection of the extracts caused no sustained depression in blood pressure. In fact, in some instances rises in blood pressure occurred. These results are good evidence for the nonexistence of a toxic material in traumatized tissue. Further controlled investigations of this type will place the burden of proof on the school of thought that supports the toxemia theory of trau-

1. Cannon, W. B.: Traumatic Shock, New York, D. Appleton & Co., 1923.

2. Cannon, W. B.: Ann. Surg. **100**: 704 (Oct.) 1934.
3. Cannon, W. B., and Bayliss, W. M.: Report on Shock Commission, Medical Research Council, no. 26, pp. 19 and 27, 1919.
4. Collip, J. B.: J. Physiol. **66**: 416 (Dec.) 1928.
5. Roome, N. W., and Wilson, Harwell: Experimental Shock, Arch. Surg. **31**: 361 (Sept.) 1935.

matic shock. In addition, they should do much to clarify other experiments that have been conducted to determine the possible functional rôle of a toxic factor in certain traumatic conditions.

VITAMIN D IN COMMON FOODS

The sources of vitamin D or vitamin D effects are now so easily available and so widely distributed that the question of an adequate supply would hardly seem to concern greatly any average American. Nevertheless, pediatricians who see constantly the effects of an inadequate supply of vitamin D are inclined to deprecate the manner in which vitamin D is made available and in which it is used by the vast majority of people.

The child is taught in school that the most potent food sources of vitamin D are egg yolk and butter, yet actually there is little vitamin D in these common foods. The amounts present in egg yolk and butter vary as much as 500 per cent during the year. A recent survey of the topic by Coffin [1] points out the erroneous conceptions that prevail in the minds of many parents. There is some belief that the fresh vegetables provide adequate vitamin D, yet the vegetables contain but little vitamin D at the time of picking and Bills has shown that even a slight amount of artificial overirradiation of ergosterol will destroy acquired potency after a maximum vitamin D potency is reached. One difficulty lies in our methods of testing for the presence of vitamin D. Biologic methods in the past have not been as accurate as they are today. No doubt many a record appeared in the periodical literature that is not sustained by reinvestigation with more modern methods. Mr. Coffin has duplicated some of the earlier work with the various vegetables and other foods. His results indicate that, with the exception of egg yolk and butter, none of the ordinary foods contain any substantial amount of vitamin D. By the use of eggs and butter alone, any attempt to obtain a daily protective amount of this vitamin would probably lead to digestive disturbances if not to an unbalanced diet.

Modern man has greatly changed the conditions of his environment. Few human beings in large Northern cities obtain any substantial amount of sunshine except possibly in summer. The main source of vitamin D must therefore come to the human being from exposure to ultraviolet rays, from natural sources of this vitamin or from extra vitamin D obtained through materials that have been enhanced in their vitamin D potency. Nutritional authorities believe that man has been deprived of the vitamin D that he used to get by natural methods and that insistence on an additional source of this vitamin will be for the benefit of mankind. Certainly, so far as the growing child is concerned, some additional vitamin D is necessary everywhere.

Quite recently the thought that an oversupply of vitamin D would be developed in this manner and that the eventual effects of this oversupply would be toxic has been reemphasized in various places. Experiments along these lines thus far reported do not warrant this belief. Clouse reported as long ago as 1932 that disastrous results from an oversupply of vitamin D did not occur in animals until the oversupply had reached at least 25,000 to 50,000 times the minimum antirachitic dose. Of course, figures are not yet available on the effects of long continued large doses for human beings. Presumably the human body possesses wide factors of safety in relationship to the assimilation of these essential substances. Thus far the evidence seems to indicate not only for vitamin D but for all the vitamins more harm in the direction of an insufficiency than in relationship to an oversupply.

1. Coffin, Joseph: The Lack of Vitamin D in Common Foods, J. Am. Dietet. A. **11**: 119 (July) 1935.

Current Comment

ADDICTION TO ENDOCRINE GLAND PRODUCTS

After careful consideration of the available terms, Patterson [1] feels that, with the possible exception of the French word "manie," addiction most nearly expresses some unusual manifestations that may result from the continued use of certain endocrine gland extracts. Though most of the cases of the misuse of endocrine gland extracts quoted by him are properly an "abuse," some are comparable in their psychologic content with instances of overmastery by morphine, diacetylmorphine and alcohol, for which the term "addiction" is used. In the use of thyroid gland products, which he considers first, instances of ill effects from prolonged or poorly supervised administration are numerous. The development of symptoms of hyperthyroidism is the most common of these complications. The especial danger of this is in prolonged self administration, usually aimed at the treatment of obesity. There are, however, cases in which the psychologic factor is the predominant influence. The effect of thyroid extract is to speed up metabolism and rate of energy transformation in the body, and it is easily understandable that there should be a certain tonic effect or increased feeling of well being. This appeared to be the factor influencing three of the author's patients in whom toxic symptoms of overdosage were produced. The frequency of misuse of adrenal extracts is in some ways more important because of the injection route of administration of the latter. Thus a person having asthma may develop the habit of taking large amounts of epinephrine at frequent intervals, even though he experiences little relief from the drug. The periodicity of the injections, moreover, is often striking. Also relatively small doses may be effective if given by the physician, but larger ones administered by the patient or his family may fail to give relief. A number of cases are cited to support the hypothesis that psychic factors may be largely responsible for the habitual use of epinephrine. Among them is that of

1. Patterson, S. W.: Addiction to Endocrine Gland Extracts, Brit. M. J. **2**: 442 (Sept. 7) 1935.

an asthmatic patient who, when deprived of his injection, committed suicide. Another case is cited in which the patient had become accustomed to giving himself from 200 to 240 injections in the twenty-four hours of the serum of Heckel, which contains epinephrine. The fear of an attack was enough to call for the injection, and the need for injection survived the cure of the asthma.[2] Patterson believes that physicians should be fully aware of the psychologic state and often neurotic condition of their asthmatic patients and should supervise and keep control of the treatment and instruct their patients in cleanliness and technic of injection. In the case of the gonads, the psychologic repercussions have been noted in the literature of the world from the earliest times. The attempt to regain youth has always been a favorite wish of mankind. But the knowledge of failure or lack of manly or womanly potentiality may have far-reaching psychologic effects. Such was apparently the situation in a case cited, that of a woman who for years had weekly injections of an ovarian extract. This she was accustomed to give herself, she was miserable without it, and substitution by other newer and presumably more potent extracts was stated by the patient to have no useful effect. Over the observation period of eight years she continued weekly injections but did not increase the dose or show any tendency to use other drugs. In other respects she seemed normal. The tendency to habit formation is on the whole a relatively rare complication of glandular therapy and may at times involve more the effect of the needle puncture than that of the drug. It is, however, the duty of medical men prescribing glandular extracts to exercise proper supervision and control of their use by patients. Physicians should be aware of the possible dangers so that the drug may be stopped at once if ill effects arise.

Association News

RADIO BROADCASTS

The American Medical Association broadcasts over the Blue network and certain additional stations of the National Broadcasting Company at 5 p. m. eastern standard time (4 o'clock central standard time, 3 o'clock mountain time, 2 o'clock Pacific time) each Tuesday, presenting a dramatized program with incidental music under the general theme of "Medical Emergencies and How They Are Met." The title of the program is "Your Health." The program is recognizable by a musical salutation through which the voice of the announcer offers a toast: "Ladies and gentlemen, your health!" The theme of the program is repeated each week in the opening announcement, which informs the listener that the same medical knowledge and the same doctors that are mobilized for the meeting of grave medical emergencies are available in every community, day and night, for the promotion of the health of the people. Each program will include a brief talk dealing with the central theme of the individual broadcast.

The next three programs are as follows:

October 29. Poisonous Plants and Animals, W. W. Bauer, M.D.
November 5. Hemorrhage, Morris Fishbein, M.D.
November 12. Infection, Morris Fishbein, M.D.

This program is broadcast also on the short waves through KDKA, Pittsburgh, over station W8XK, 11,870 and 12,210 kilocycles.

2. Flandin, M.: Bull. et mém. Soc. méd. d. hôp. de Paris **46**: 116 (Feb. 3) 1930. (This case seems almost unbelievable but is correctly quoted.)

Medical News

(PHYSICIANS WILL CONFER A FAVOR BY SENDING FOR THIS DEPARTMENT ITEMS OF NEWS OF MORE OR LESS GENERAL INTEREST: SUCH AS RELATE TO SOCIETY ACTIVITIES, NEW HOSPITALS, EDUCATION, PUBLIC HEALTH, ETC.)

ALABAMA

Society News.—Dr. H. Earle Conwell, Fairfield, presented a paper recently on the treatment of fractures before the Franklin County Medical Society in Russellville. Dr. Conwell addressed a joint meeting of the Elmore and Tallapoosa county medical societies at Camp Dixie on Lake Martin, recently, on "Fractures About the Elbow, Especially in Children."

Personal.—Dr. Edwin P. Moon has recently been appointed state physician for prison number 1 and the tuberculosis hospital in Wetumpka.——Dr. Hampton E. Barker, Haleyville, has been named health officer of Bullock County, succeeding Dr. Leslie G. Cole, who has resigned to accept a similar position in Wetumpka.——Dr. Julius E. Dunn, Covington, Ky., has been appointed health officer of DeKalb County.

CONNECTICUT

Changes in Health Officers.—Dr. Samuel Green has been appointed health officer of Southbury and Dr. Julian G. Ely, health officer of Lyme, succeeding Dr. Matthew Griswold. Dr. Jacob Edward Waldman, Middletown, has been named town health officer of Haddam.

Society News.—A special dinner meeting of the radiologic section of the Connecticut State Medical Society was held September 18, during the eleventh clinical congress of the society. By-laws were discussed for adoption and other matters pertaining to the cooperation of the radiologists in the state were presented. Dr. Charles W. Perkins, Norwalk, is chairman of the section, and Dr. Max Climan, Hartford, secretary.

ILLINOIS

Personal.—Dr. Francis N. Orr has resigned from the staff of the Lincoln State School and Colony, Lincoln, after four years' service, to engage in private practice.

Society News.—Dr. Edward Lee Dorsett, St. Louis, addressed the Madison County Medical Society at Madison, October 4, on "Conservative Treatment of Eclampsia."——At a meeting of the La Salle County Medical Society at Ottawa, September 26, speakers were Drs. Edward L. Cornell, Chicago, on "Fundamentals of Obstetrics"; Charles E. Galloway, Evanston, "Timely Pointers on Obstetrical Delivery," and Irving F. Stein, Chicago, "Lipiodol as an Aid in Gynecologic Diagnosis."

Chicago

Personal.—Dr. Jacob P. Greenhill has been elected head of the department of obstetrics at the American Hospital.——Dr. Leo M. Czaja has been appointed general superintendent of the Municipal Tuberculosis Sanitarium.

Lead Poisoning from Burning Storage Batteries.—Lead fumes from a kitchen stove where storage battery casings were being burned as fuel caused the death of two children and the serious illness of five others in a family of eleven persons, the Chicago *Tribune* reported October 20.

Pathologic Conferences.—Dr. Richard Jaffe opened a series of pathologic conferences at the Cook County morgue, October 18, on current material from the children's department of Cook County Hospital. Conferences are scheduled for November 15, December 13, January 10, February 7, March 6, April 3, May 1 and May 29.

Hospital News.—The Iroquois Memorial Hospital was opened, October 17, as a branch of the Municipal Tuberculosis Sanitarium; it will be devoted exclusively to collapse therapy of tuberculosis. The hospital, which has been closed since January 1 for lack of funds, was built in 1910 as a memorial to those persons who died in the Iroquois Theater fire in December 1903.

Society News.—Speakers before the Chicago Neurological Society, October 17, included Drs. Isidore Finkelman and William Mary Stephens, Elgin, Ill., on "Heat Regulation in Chronic Encephalitis."——The staff of the Sarah Morris Hospital for Children of the Michael Reese Hospital presented the program of a clinical meeting before the Chicago Pediatric Society, October 15.——At a meeting of the Chicago Society of Allergy,

October 21, Dr. Samuel M. Feinberg discussed "A Study of Molds and Their Relation to Allergic Diseases," and Dr. Francis L. Foran opened a discussion on the 1935 hay fever season.

Fund for Dental Research.—An anonymous contribution has made possible the establishment of a foundation for dental research in the Chicago College of Dental Surgery, the dental division of Loyola University, which will have available annually $25,000 as a minimum. The major part of the work will be carried on in the laboratories of the dental school, while another part will be undertaken at the John McCormick Institute for Infectious Diseases. Some of the problems to be investigated will be a study of the bacteriology and histopathology of dental caries and the diseases that involve the supporting structures of the teeth as well as their effects on general health.

INDIANA

Personal.—Dr. Joseph W. Strayer, for several years resident physician at the Boehne Tuberculosis Hospital, Evansville, has been appointed superintendent of the Smith-Esteb Memorial Tuberculosis Hospital, Richmond.——Dr. Werner W. Duemling, Fort Wayne, has been made chief physician of the city's public schools, succeeding Dr. Charles R. Dancer, who held the position twenty-six years.

Society News.—Dr. John MacMillan Townsend, Louisville, Ky., addressed the Wayne-Union Counties Medical Society in Richmond, September 19, on recent advances in urology.—— At a meeting of the Floyd County Medical Society in New Albany, September 26, Dr. Charles P. Emerson, Indianapolis, presented a paper entitled "Why So Much Surgery in America?"——Dr. Frazier N. Cloyd, Danville, Ill., spoke on "Care of Common Fractures of the Arm and Leg" before the Fountain-Warren Counties Medical Society in Covington, October 3. ——Dr. Burrill B. Crohn, New York, conducted a gastroenterologic clinic before the Indianapolis Medical Society, October 15, in the auditorium of the University of Indiana School of Medicine; in the evening he spoke at the Athenaeum on "Differential Diagnosis of Diarrheas." —— Dr. Edward A. Oliver, Chicago, discussed treatment of cancerous lesions of the skin before the Elkhart County Medical Society, Elkhart, October 3.

IOWA

Rocky Mountain Spotted Fever. — Six cases of Rocky Mountain spotted fever have been reported in Iowa thus far in 1935, all in the southern part of the state. The two cases reported in September were from Lee and Linn counties; other counties involved are Poweshiek and Union. No cases were reported during August.

Society News.—Speakers before the Boone-Story County medical societies in Ames, September 18, included Drs. Philip H. Kreuscher, Chicago, on "Treatment of Fracture of the Larger Joints," and Newton D. Smith, Rochester, Minn., "Fistulae and Hemorrhoids."——The Calhoun County Medical Society was addressed in Rockwell City, September 26, by Dr. Johann Erwin von Graff, Des Moines, on "Miscarriages, Diagnosis, Complications and Treatment."——The Cerro Gordo County Medical Society approved the establishment of a county tuberculosis society at its meeting in Mason City, September 10. Dr. Elexious T. Bell, Minneapolis, spoke on "Clinical Diagnosis of Tumors of the Breast."——Dr. William Wayne Babcock, Philadelphia, addressed the Johnson County Medical Society in Iowa City, October 5, on "Common Errors in Surgical Practice."——At a meeting of the Wapello County Medical Society in Ottumwa, September 17, Dr. William C. Newell, Ottumwa, discussed "Diseases of the Liver and Biliary Tract." ——Dr. Edward Jackson, Denver, will discuss "Practical Ophthalmology for Physicians and Surgeons" before the Linn County Medical Society, November 14, in Cedar Rapids.

KANSAS

Physicians Needed in Civilian Conservation Corps.—There are a few vacancies on the medical staff of the Kansas District, Civilian Conservation Corps. All physicians who are interested are requested to write the surgeon of the district, Fort Riley, for details.

Society News.—Dr. Howard L. Alt, Chicago, addressed the Shawnee County Medical Society in Topeka, October 6, on "Origin and Morphology of Blood Cells with Application to Disease." —— The Sedgwick County Medical Society was addressed, October 1, among others, by Drs. Wilbur G. Gillett, Wichita, on "Foreign Protein in the Treatment of the Eye," and Willard J. Kiser, Wichita, on "Pulmonary Embolism." —— At a meeting of the Golden Belt Medical Society in McPherson, October 3, speakers included Dr. Noble E. Melencamp, Dodge City, on "Changing Concepts Regarding the Endometrium and Their Significance." —— The Tri-County Medical Society, composed of Sumner and Cowley counties, Kan., and Kay County, Okla., was addressed in Winfield, September 19, by Dr. Howard H. Bradshaw, Boston, among others, on "Anesthesia and Surgery as Applied to Collapse Therapy in Tuberculosis."

KENTUCKY

Changes in Health Departments.—Dr. Fred W. Caudill, Georgetown, has resigned as health officer of Scott County to become epidemiologist of the state health department. Dr. Robert J. Griffin, Louisville, succeeds Dr. Caudill in Scott County. A new health unit has been established in Lyon County with Dr. Neale M. Atkins, Eddyville, in charge. Dr. Charles M. Moore, Glasgow, who has been health officer of Barren County, has been appointed city health officer of Lexington.

MARYLAND

Personal.—Dr. Hosea W. McAdoo, superintendent of the Springfield State Hospital, Sykesville, has resigned to accept a position at Cambridge, Mass.

Dr. Gunn to Give Dohme Lectures.—Dr. James A. Gunn, professor of pharmacology and director of the Nuffield Institute of Medical Research, University of Oxford, will deliver the Dohme Lectures at Johns Hopkins School of Medicine, Baltimore, November 6-8. The titles of the lectures are: "Pharmacologic and Therapeutic Properties of the Harmala Alkaloids and Their Derivatives," "Reactions of Uterine Muscle" and "Pharmacologic Syndromes."

Society News.—The first fall meeting of the Baltimore City Medical Society, October 4, was designated "house officers' night," with the following speakers: Drs. Thomas M. Brown, Johns Hopkins Hospital, "Epidemic Meningococcus Meningitis"; Harry C. Hull, University Hospital, "Primary Carcinoma of the Small Intestine"; Philip D. Flynn, Mercy Hospital, "A Case Report of Rocky Mountain Spotted Fever"; Joseph I. Ostergren, Kernan's Hospital, "Xanthomatosis, Schüller-Christian Type," and Harold C. Dix, Maryland General Hospital, "End Results of Complete Hysterectomy."

Visit to Home of First Medical Graduate.—The memory of John Archer, M.B., who had the distinction of being the first graduate of an American medical school, was honored during the semiannual meeting of the Medical and Chirurgical Faculty of Maryland in Bel Air, Harford County, October 10. About 200 members of the faculty visited Medical Hall, seven miles from Bel Air, the birthplace and home of Dr. Archer, and Churchville Cemetery, where Dr. Armfield F. Van Bibber, a descendant of the physician, placed a wreath on his grave. Dr. Archer was born May 5, 1741, near the present village of Churchville. He received the degree of bachelor of medicine in 1768 from the University of Pennsylvania Medical Department, returning to Harford County in July 1769, where he practiced forty years. During the Revolution he was a member of the local committee from November 1774 and in December of the same year was captain of a militia company, becoming major in January 1776. In August of the same year he was a member of the convention that framed the Maryland constitution and bill of rights. He was one of the founders of the Medical and Chirurgical Faculty of Maryland in 1799 and served as a member of Congress from 1801 to 1807. He died in 1810.

MASSACHUSETTS

Personal.—Dr. Henry M. Pollock, superintendent, Massachusetts Memorial Hospitals, has been reappointed associate commissioner of the state department of mental diseases.—— Dr. James G. Bruce has been appointed surgeon for the police and fire departments of Springfield.

Changes in Medical Examiners.—Dr. John A. Huffmire, Huntington, has been appointed associate medical examiner of the second Hampshire District. Dr. John V. Gallagher, Milford, has been named medical examiner of the sixth Worcester District, to succeed the late Dr. George F. Curley.

Dinner in Honor of Dr. Edsall.—The Harvard Medical School Alumni Association held a dinner in honor of Dr. David L. Edsall, at the Harvard Club of Boston, October 23. Dr. Edsall recently retired as dean of Harvard Medical School. James B. Conant, LL.D., president of Harvard University, presided at the dinner, and speakers included A. Lawrence Lowell, LL.D., president emeritus of Harvard, and Dr. Charles S. Burwell, dean of the medical school. At a meeting preceding the dinner, addresses on "The Development of Medical

Education in the United States Since the World War" were made by Walter A. Jessup, LL.D., New York, president, Carnegie Foundation for the Advancement of Teaching; Dr. Eugene F. Du Bois, professor of medicine, Cornell University Medical School, New York, and Dr. Lawrence J. Henderson, professor of biological chemistry at Harvard. Dr. Walter B. Cannon, George Higginson professor of physiology at Harvard, presided at this meeting.

Medical School Lectures.—Dr. Walter C. Alvarez, Rochester, Minn., addressed the William Harvey Society of Tufts College Medical School, October 25, on "Functional Digestive Disorders." Other meetings of the society will be addressed by the following:

Dr. Harold E. B. Pardee, assistant professor of clinical medicine, Cornell University Medical School, New York, Arteriosclerotic Heart Disease, November 8.

Dr. Martin H. Dawson, assistant professor of medicine, Columbia University College of Physicians and Surgeons, New York, Rheumatoid Arthritis and Osteo-Arthritis, December 13.

Dr. Hiram Houston Merritt, instructor in neurology, Harvard Medical School, Boston, Syphilis of the Nervous System, January 10.

Dr. Luther Emmett Holt Jr., associate professor of pediatrics, Johns Hopkins University School of Medicine, Baltimore, Significance of Fats in Nutrition, February 14.

Dr. Alexander Lambert, formerly professor of clinical medicine, Cornell University Medical School, New York, Therapeutics of Drug Habits, March 13.

Dr. Elliott C. Cutler, Moseley professor of surgery, Harvard Medical School, Boston, War Surgery, April 10.

MICHIGAN

Antimeningococcus Serum Available.—The Michigan Department of Health announces that therapeutic doses of antimeningococcus serum are available to any physician on request. The new product will be handled by the regular distributors of biologic products manufactured by the department. The serum is put up in vials of 20 cc. each.

Dr. Stapleton Named Acting Dean.—At the regular meeting of the Detroit Board of Education, August 27, it was decided not to appoint a dean of Wayne University College of Medicine at present; Dr. William J. Stapleton, who was appointed assistant dean at the June meeting of the board, was named acting dean until the end of the year.

Memorial to Dr. Harison.—A tablet is to be mounted in a public park in Sault Ste. Marie in honor of the late Dr. Beverly Drake Harison, with the following inscription:

BEVERLY DRAKE HARISON
1855-1924

Prominent Sault Ste. Marie physician. Foremost in Michigan medical registration. One of the founders of the Upper Peninsula Medical Society—President of the Michigan Medical Society 1904.

Personal.—Dr. Clarence H. Westgate, Morenci, has been appointed medical director of the Lenawee County Tuberculosis Sanatorium, Adrian, succeeding Dr. Wellington B. Huntley, now chief of the medical staff at the Southern Michigan Prison at Jackson.——Dr. Louis A. Schwartz, Detroit, has been appointed consulting psychiatrist for the Social Service Federation of Toledo, Ohio.——Dr. George W. Robinson, Detroit, has been appointed chief of the obstetrical department of St. Mary's Hospital, succeeding the late Dr. James W. Cunningham.——At the opening exercises of the University of Michigan Medical School, Ann Arbor, the honorary degree of master of arts was conferred on Dr. Andrew P. Biddle, Detroit, "in recognition of a life devoted to the advancement of education and ethics in the medical profession."——Dr. Lowell S. Selling, Detroit, has been named psychiatrist in the recorder's court, succeeding the late Dr. Isaac L. Polozker.

MINNESOTA

Personal.—Honorary membership in the Minneapolis Clinical Club was conferred on Dr. Adolph M. Hanson, Faribault, at a meeting, October 10; Dr. Hanson gave an address entitled "Some Aspects of the Thymus and Pineal Problems."——Dr. Clifford T. McEnaney, Owatonna, has been named physician and surgeon for the Minnesota State Public School, succeeding Dr. Allan B. Stewart, who resigned after many years' service.

Violation of Basic Science Law.—Arthur N. Alexander, St. Paul, a licensed chiropodist, pleaded guilty, September 19, to a charge of practicing healing without a basic science certificate. Evidence was produced to show that Alexander had performed an abortion. Because he was unable to raise $5,000 bail, he was confined in the Ramsey County jail. Following his plea of guilty he surrendered his chiropody license. In view of this and of the fact that he had been in jail since his arrest, Judge Boerner imposed a suspended sentence of one year.

Society News.—Dr. Melvin S. Henderson, Rochester, presented a case of atrophy of both bones of the forearm before the Minnesota Academy of Medicine, October 9, in Minneapolis; Dr. Ernest M. Hammes, St. Paul, spoke on "Spinal Cord Injuries," and Dr. Franklin R. Wright, Minneapolis, reported a case of "Reflex Urinal Frequency."——Dr. Hobart A. Reimann discussed "Progress in Infectious Diseases" before the Hennepin County Medical Society, Minneapolis, October 16, and Dr. Frank H. Krusen, Rochester, spoke, October 23, on "Present Trends in Physical Therapy." The society will be addressed, October 30, by Drs. Nathaniel H. Lufkin and Henry W. Quist on "Island Tumors of the Pancreas" and "Acute Intestinal Obstruction," respectively. Dr. William Boyd, Winnipeg, will speak, November 7, on "The Decalcifying Diseases of Bone."——At a meeting of the Scott-Carver County Medical Society, September 10, at Mudbaden, speakers were Drs. Owen H. Wangensteen, Minneapolis, on acute conditions of the abdomen; Frederic E. B. Foley, St. Paul, improved operation for ureteral stone, and John F. Briggs, St. Paul, achlorhydric anemia.

MISSISSIPPI

Society News.—The Adams County Medical Society was addressed recently by Dr. Jacob S. Ullman, Natchez, on "Menstruation and the Hormones."——The Jones County Medical Society was organized at the South Mississippi Charity Hospital in Laurel, recently, with Dr. Alcus J. Carter, Ellisville, president; Dr. James B. Jarvis, Laurel, vice president, and Dr. Eugene A. Bush, Laurel, secretary.

MISSOURI

University News.—Dr. Theodore L. Waddle, associate director of the St. Landry Parish Health Unit, Louisiana, has been appointed assistant professor of physiology at the University of Missouri School of Medicine, Columbia, to succeed Dr. Robert W. Siddle. Dr. Robert M. Moore, instructor in bacteriology and pathology, University of Tennessee School of Medicine, has been named assistant professor of pathology to succeed Dr. Clarence C. Pflaum.

New Home for County Society.—New headquarters for the Jackson County Medical Society will be included in a building to be erected soon on "hospital hill" in connection with the Kansas City General Hospital, between the present main structure and the isolation hospital. An auditorium and adjoining small rooms for committee meetings will be at the disposal of the society. The new quarters will be maintained by the city in recognition of the services of the profession to the community.

Society News.—At a meeting of the St. Louis Medical Society, October 1, Dr. Sigismund S. Goldwater, New York, discussed "Handicapped Hospitals—Public and Private" and Dr. Nathaniel W. Faxon, Boston, "Voluntary Hospital Insurance." The society devoted its meeting, September 17, to a consideration of anterior poliomyelitis. Drs. Emanuel Sigoloff discussed epidemiology; Jean V. Cooke, diagnosis and treatment of the acute stage, and Clarence H. Crego Jr., the orthopedic treatment. At this meeting the name of the code and contract board was changed to the medical economics board by unanimous vote.——Dr. Louis L. Tureen, St. Louis, discussed "Pathology of Air Embolism," among other speakers before the Trudeau Club of St. Louis, October 3.——Dr. Lynn M. Garner, Tuscumbia, was chosen president-elect of the Missouri Public Health Association at its joint session with the Missouri Tuberculosis Association in Kansas City, September 5-7; Dr. Joseph F. Bredeck, St. Louis, is president. Dr. Irl B. Krause, Jefferson City, was reelected president of the Missouri Tuberculosis Association.

NEW JERSEY

Society News.—The program of the Bergen County Medical Society, October 8, in Hackensack, was devoted to ophthalmologic subjects of interest to the general practitioner; speakers were Drs. Charles Littwin, Englewood, Samuel T. Hubbard, Raynold N. Berke and Arcangelo Liva.——Dr. Cornelius P. Rhoads, New York, addressed the Hudson County Medical Society, Jersey City, October 1, on "Treatment of Anemias."——Dr. Bernard I. Comroe, Philadelphia, addressed the Gloucester County Medical Society, Woodbury, September 19, on abdominal pain.——Dr. Chevalier Jackson, Philadelphia, addressed the Burlington County Medical Society, September 12, on "Bronchoscopy as an Aid to the General Practitioner."

State Board Activities.—Within the past few months the State Board of Medical Examiners of New Jersey has reported the following activities, among others, in enforcing the medical practice act:

Michael Jaroslaski was found guilty of practicing medicine without a license.

Charles W. Remboy, an "herb doctor" of Butler, was found guilty of practicing medicine without a license and sent to jail for thirty days.

Cleon E. Shields, Newark, pleaded guilty to practicing without a license and paid the penalty. Shields claimed to improve impaired hearing by the use of a machine called "Auratone," which he said measures the capacity to hear and furnishes in correct volume the tones that are "blocked out" by the impairment.

Julian Cyzykowski, Englishtown, was found guilty of practicing without a license.

Among other offenders prosecuted were five druggists, one osteopath, two chiropractors, two naturopaths and two masseurs.

NEW YORK

Milk-Borne Bacillary Dysentery. — One hundred and thirty-one persons in a village of 500 were attacked by bacillary dysentery between August 25 and September 15 in an outbreak that was traced to the use of raw milk. The suspected dairy supplied about three fourths of the raw milk used in the village. Among 339 users of both the milk and the village water supply, ninety-three became ill, and the other cases were among those using the milk but not the water and those which could be considered contact cases. The original source of the infection is not known, according to *Health News*. Among twelve persons on the dairy farm, four were ill, but their illness occurred during the outbreak and not before it. In addition, dysentery bacilli were isolated from two other members of the household, one of whom was found to be a healthy carrier.

New York City

Personal.—Dr. John E. Gordon, formerly epidemiologist of the Detroit Department of Health, who joined the staff of the Rockefeller Foundation in 1934, has gone to Rumania, where with a staff of laboratory and field workers he will make a study of scarlet fever.

Society News.—Dr. William Goldring gave a Friday afternoon lecture before the Medical Society of the County of Queens, Forest Hills, October 4, on "Clinical Aspects of Nephritis and Hypertension." Dr. Benjamin M. Bernstein spoke, October 18, on "Ulcerative Colitis." —— Dr. Henri Coutard, chief of the department of roentgen therapy for cancer, Curie Institute of the University of Paris, addressed the New York Roentgen Society and the section of otolaryngology of the New York Academy of Medicine, October 16, on "Roentgen Therapy of Epitheliomas of the Tonsillar Region, Hypopharynx and Larynx."——Drs. Edward W. Peterson and Louise H. Meeker addressed the New York Surgical Society, October 9, on "Tumors of the Carotid Body."——Dr. Reuben Ottenberg addressed the Bronx Pathological Society, October 15, on "Sepsis Arising from Trauma."——Drs. Meyer Rosensohn and Samuel H. Geist will address the Bronx Gynecological and Obstetrical Society, October 28, on "Toxemias of Pregnancy" and "Gynecological Surgery," respectively. —— Drs. Edward J. O'Brien, Detroit, and Foster Murray, Brooklyn, addressed the Medical Society of the County of Kings, October 15, on "Design for Management of Pulmonary Tuberculosis."

Dr. Sabin to Receive Bryn Mawr Award.—Dr. Florence R. Sabin, member of the Rockefeller Institute for Medical Research, has been announced as the third recipient of the M. Carey Thomas Prize of $5,000, which is given at intervals by Bryn Mawr College, Bryn Mawr, Pa., to an American woman in recognition of eminent achievement. The presentation will be made November 2 at the college as the climax of a two day program celebrating the fiftieth anniversary of its founding. The award was founded by the alumnae association in honor of Miss Thomas and presented to her in 1922, when she retired as president of Bryn Mawr. The second presentation was to the late Jane Addams of Hull House, Chicago, in 1931. Dr. Sabin, a native of Colorado, took her medical degree at Johns Hopkins University School of Medicine, Baltimore, in 1900, and was a member of the faculty from 1902 to 1925, when she went to the Rockefeller Institute. She was professor of anatomy for twelve years and professor of histology for the next eight years. She was the first woman to be admitted to and to graduate from Johns Hopkins, the first woman intern at Johns Hopkins Hospital, the first woman member of the National Academy of Sciences and the first woman member of the Rockefeller Institute. She has received various honorary degrees, and in 1929 was awarded the *Pictorial Review* Prize of $5,000 for distinguished achievement.

NORTH CAROLINA

Personal. — Dr. James Allen Whitaker, Mount Airy, has resigned as health officer of Surry County to take charge of the health department of Rocky Mount. —— Dr. Clarence H. White, Kenansville, has been appointed district health officer in charge of Avery, Yancey and Watauga counties.

Campaign Against Hookworm.—The International Health Division of the Rockefeller Foundation, New York, has made available $12,000 to the state board of health to finance a campaign against hookworm. Dr. Alvin E. Keller, Nashville, Tenn., will direct the work, which will include a survey and campaign of prevention and prophylaxis.

OHIO

Changes at University of Cincinnati.—Dr. John H. Foulger, associate professor of pharmacology at the University of Cincinnati College of Medicine, has resigned to join the staff of the Haskell Laboratory for Toxicological Research, Wilmington, Del. Dr. Norman A. David, assistant professor of pharmacology, University of West Virginia School of Medicine for the past three years, has been appointed assistant professor in the department at Cincinnati.

Personal.—Dr. Carl Watson, Findlay, has been appointed works progress administrator for Ohio.——Dr. James F. Busby, Northville, Mich., has been appointed superintendent of the Licking County Tuberculosis Sanatorium, Newark, succeeding Dr. Donald E. Yochem, who resigned to enter private practice. ——Dr. Harry W. Wertz, Montpelier, was appointed health officer of Williams County to succeed the late Dr. William A. Held, West Unity.——Dr. Will M. Hoyt, Hillboro, has been appointed health officer of Highland County to succeed Dr. John L. Walker.

Henry Hoffman Arrested.—Dayton, Ohio, newspapers of October 9 report that Henry Hoffman of Rosewood, Ohio, was arrested at Tippecanoe City, October 9, on an indictment charging him with practicing medicine illegally. A bond of $100, with one Christopher Metzgar as surety, was furnished by Hoffman and he was released. It is reported that Hoffman accepted a fee for medical service and also prescribed certain medicines, although the man is not a physician. The paper states, also, that Hoffman was convicted on the same charge at Eaton a year ago and fined $50. It is claimed that in writing prescriptions for medicines, Hoffman makes a prescription in such a form that it cannot be filled at a drug store, but the patient—or victim—must go to an alleged agent of Hoffman. According to the report, the agent selling Hoffman's nostrums in Tippecanoe City is Metzgar, the man who put up the $100 bond. The files of the Bureau of Investigation of the American Medical Association contain a clipping from a Greenville, Ohio, newspaper of June 1934, reporting plans for a retrial in the state's case against one Henry Hoffman of Verona, an alleged faith healer.

OKLAHOMA

Personal. — Dr. John T. Looney, Tishomingo, has been appointed health officer of Johnston County. —— Dr. Max H. Grow has been appointed physician to the Oklahoma Agricultural and Mechanical College at Stillwater.

Society News.—At a meeting of the Southern Oklahoma Medical Association at the Central State Hospital, Norman, September 3, speakers included Drs. Carl T. Steen of the hospital staff, on "Sterilization as Applied to the Insane"; Wann Langston and Bert E. Mulvey, Oklahoma City, "Symposium on the Heart and Interpretation of the Electrocardiogram," and Ray M. Balyeat, Oklahoma City, "Use of Iodized Oil in Treatment of Asthma."——Dr. Samuel R. Cunningham, Oklahoma City, addressed the Cleveland County Medical Society, Norman, October 1, on "Fractures of the Knee and Elbow: Their Diagnosis and Treatment."——Dr. E. Rankin Denny, Tulsa, addressed the Tulsa County Medical Society, October 14, on "Significance of Blood Cholesterol Determinations in Hypometabolism."

PENNSYLVANIA

Society News.—A graduate conference was held at Wilkes-Barre General Hospital recently with clinics in the morning anl lectures in the afternoon. Guest speakers were Drs. George W. Crile, Cleveland; Emanuel Libman, New York, and Emil Novak, Baltimore.——Dr. Francis C. Grant, Philadelphia, addressed the Cambria County Medical Society, Johnstown, October 10, on "Diagnosis and Surgical Treatment of Brain Tumors."——Dr. Milton H. Cohen, Lewistown, addressed the Mifflin County Medical Society, Lewistown, September 5,

on "Female Sex Hormones and Their Clinical Application."——Drs. Arthur M. Shipley and Maurice C. Pincoffs, Baltimore, conducted the annual fall clinic of the Fayette County Medical Society, Uniontown, October 21.

Philadelphia

Faculty Changes at the University.—Dr. Carl Edwin Bachman, Montreal, Canada, has been appointed assistant professor of obstetrics and gynecology at the University of Pennsylvania School of Medicine. Dr. Bachman is a native of Pennsylvania and a graduate of the university. Drs. Edward H. Campbell and Harry P. Schenck have been promoted to be assistant professors of otolaryngology.

Society News.—Among speakers at a meeting of the Philadelphia Academy of Surgery, October 7, were Drs. John O. Bower, Newlin F. Paxson and Harold A. K. Mengle, who presented "A Preliminary Report of the Presence of Clostridium Welchii Antitoxin in the Blood of Patients Who Have Recovered from Peritonitis, the Result of a Perforated Appendix."

Opening of Woman's College.—Dr. Chevalier Jackson, recently elected president of the Woman's Medical College of Pennsylvania, and Dr. Louise Pearce, New York, made addresses at the opening of the new session, September 26. The following appointments were announced:

Dr. Esther M. Greisheimer, formerly associate professor of physiology, University of Minnesota, professor of physiology.

Dr. Hartwig Kuhlenbeck, formerly professor of anatomy at the University of Breslau, Germany, acting professor of anatomy.

Ben King Harned, Ph.D., formerly chief of the division of pharmacology, University of Tennessee College of Medicine, Memphis, professor of pharmacology.

Versa Viola Cole, Ph.D., formerly research assistant, Ohio State University College of Medicine, Columbus, assistant professor of pharmacology.

Marion Fay, Ph.D., formerly lecturer of chemistry, University of Texas School of Medicine, professor of physiological chemistry.

Marie Andersch, Ph.D., formerly of the pathological chemistry laboratory, University Hospitals, State University of Iowa, assistant professor of physiological chemistry.

Dr. Mary Bickings Thornton, formerly part time professor of anatomy at the college, was made professor emeritus. To improve the facilities of the library a full time librarian has been appointed. Dr. Jackson succeeds Mrs. James Starr, who resigned as president to devote more time to the financial problems of the institution as chairman of the executive committee.

SOUTH CAROLINA

Society News.—At the annual meeting of the Seventh District Medical Association in Manning, September 19, speakers included Drs. Verling K. Hart and George Aubrey Hawes, Charlotte, N. C., on "Bronchoscopic and Clinical Observations in Bronchial Obstruction and Bronchiectasis" and "Urologic Symptoms Resulting from Pathologic Conditions of the Female Urethra," respectively, and Edgar F. Fincher, Atlanta, "Tumors of the Brain."——Drs. George T. McCutchen and Lucius E. Madden addressed the Columbia Medical Society, September 9, on "Hyperinsulinism" and "Pulmonary Aneurysm," respectively.

TENNESSEE

Infant Mortality.—In an effort to reduce the high infant mortality rate in Memphis, the city health department has increased the number of its clinics for well infants under the direction of a full time pediatrician, Dr. Beulah M. Kittrell, formerly of Knoxville. The rate of 112 per thousand live births in 1934 was the highest in the United States for cities of 30,000 population and over, it was said. For the first five months of 1934 the rate was 121.6 and for the same period of 1935 it was 94.7.

Health at Nashville.—Telegraphic reports to the U. S. Department of Commerce from eighty-six cities with a total population of 37 million, for the week ended October 12, indicate that the highest mortality rate (19.4) appears for Nashville and that the rate for the group of cities as a whole was 10.5. The mortality rate for Nashville for the corresponding week of last year was 16.5 and for the group of cities was 10.2. The annual rate for the eighty-six cities for the forty-one weeks of 1935 was 11.4 and the same rate appears for the corresponding period of the previous year. Caution should be used in the interpretation of these weekly figures, as they fluctuate widely. The fact that some cities are hospital centers for large areas outside the city limits or that they have a large Negro population may tend to increase the death rate.

Society News.—Dr. Louis Levy addressed the Memphis and Shelby County Medical Society recently on "Vincent's Angina."——Drs. Jesse B. Swafford and Edward E. Reisman addressed the Hamilton County Medical Society, Chattanooga, October 3, on "Dementia Praecox" and "Diagnostic Observations on Ectopic Pregnancy," respectively.——At a meeting of the Hardin, Lawrence, Lewis, Perry and Wayne county medical societies in Hohenwald recently, speakers were Drs. William E. Boyce, Flat Woods, on "Mechanism and Management of Normal Labor"; William R. Cate, Nashville, "Clinical Diagnosis and Significance of Irregularities of the Heart" and Ernest W. Goodpasture, Nashville, "Value of Vaccine in Poliomyelitis."——Dr. Richard A. Barr, Nashville, addressed the Gibson County Medical Society recently on "Abdominal Operations on Children."——Drs. Roy H. Ruble, Limestone, and Edward T. West, Johnson City, addressed the Washington County Medical Society, Johnson City, September 5, on "Diabetes Mellitus" and "Carcinoma of the Cervix," respectively.

VIRGINIA

New Building for Medical College.—Through an anonymous gift of $300,000 and a federal grant of $239,850, the Medical College of Virginia, Richmond, will shortly begin construction of a new outpatient clinic and laboratory building, seven stories high. The first four floors will be for clinics and the upper floors for laboratories in pathology, bacteriology, biochemistry, public health and preventive medicine. It is expected that a hospital will be built later adjoining the clinics.

Society News.—At the quarterly meeting of the Augusta County Medical Association in Staunton, recently, speakers were Drs. Lemuel Nelson K. Bell, Waynesboro, on "Medical and Surgical Problems in China"; Clarence P. Obenchain, Staunton, "Relief of Suffering During Confinement," and Kenneth Maxcy, University, "Infantile Paralysis."——Drs. Otis T. Amory and Edward L. Alexander, Newport News, addressed the Mid-Tidewater Medical Society, Gloucester Courthouse, recently, on "Indigestion" and "Antigens and Vaccines and Their Uses," respectively.——Dr. Collins D. Nofsinger, Roanoke, addressed the Montgomery, Floyd and Giles county medical societies at a joint meeting in Saint Albans, recently, on "Diagnosis and Treatment of Myocarditis."——At the quarterly meeting of the Southside Virginia Medical Association in Burkeville, September 10, speakers included Drs. Thomas F. Wheeldon, Richmond, on "Value of the Cystine Content Determination in the Treatment of Arthritis"; Wilkins J. Ozlin, South Hill, "Diagnosis in Diseases of the Chest," and Roy K. Flannagan, Richmond, "Medical Economics."

WASHINGTON

Society News.—Dr. Henry Schmitz, Chicago, addressed the King County Medical Society, October 21, on "Early Diagnosis and Indications for Treatment of Carcinoma." Dr. Alfred A. Strauss, Chicago, addressed a special meeting, October 25, on "Surgical Diathermia for Treatment of Carcinoma of the Rectum and Stomach."——At a meeting of the Klickitat County Medical Society, September 27, Dr. Dimitry V. Ogievsky, Klickitat, was elected president and Dr. Frank G. LeFor, Goldendale, secretary. A woman's auxiliary was organized with Mrs. Allen Bonebrake, Goldendale, as president. Plans were made for an active campaign for control of diphtheria and smallpox.——Dr. John C. Lyman, Walla Walla, was elected president of the Walla Walla Valley Medical Society at a meeting in Walla Walla, October 10; Rudolf A. Clemen, president of Whitman College, made an address.

WISCONSIN

Society News.—Dr. Maurice Brodie, New York, delivered the Lippitt Memorial Lecture of the Medical Society of Milwaukee County, October 11, on "Poliomyelitis: Its Transmission, Prevention and Treatment," and Dr. Walter P. Blount spoke on "After-Care of Paralytic Poliomyelitis."——Speakers at a meeting of the Ninth Councilor District Medical Society at Wisconsin Rapids recently were Drs. William F. Wilker, Iola, on "Complete Avulsion of the Scalp"; James K. Trumbo, Wausau, "The Incidence of Allergy in the Etiology of Nasal Polyps," and Norbert Enzer, Milwaukee, "Relation of Pathology to the Practice of Medicine."

State Society Awards.—The Council of the State Medical Society of Wisconsin at the annual session in Milwaukee, September 19, presented its award for distinguished service to the society, the medical profession and public health to Edward Asahel Birge, LL.D., Madison, president emeritus of the University of Wisconsin, and to Dr. Arthur Jackson Patek, Milwaukee, founder and for several years editor of the *Wisconsin Medical Journal*. Dr. Birge, now 84 years old, was for fifty years associated with the university as instructor in natural history, professor of zoology, dean of the college of arts and

sciences (1891 to 1918) and president (1918 to 1925). Dr. Patek is a former president of the Medical Society of Milwaukee County and the State Medical Society of Wisconsin and was originator and for twenty-six years secretary of the medical defense fund of the state society.

GENERAL

Examinations in Dermatology.—The American Board of Dermatology and Syphilology will hold oral examinations, May 11-12, 1936, in Kansas City, Mo., during the annual session of the American Medical Association. The written examination for Group B will be held on Saturday, March 14, in various cities throughout the country; applications for this examination should be filed before Jan. 15, 1936, with the secretary, Dr. Clarence Guy Lane, 416 Marlborough Street, Boston.

Report of National Research Council.—During the past year the National Research Council has made 116 grants totaling $50,359.50. The largest single amount, $12,600, was expended for medical sciences. Ninety-six postdoctorate fellowships have been supported at a total expenditure of $221,927.73. Among these, ten were in the medical sciences. The principal enterprises aided in the medical field were research on problems of sex, in which emphasis will now be laid on synthetic production of hormones, investigations on narcotic drugs at the universities of Michigan and Virginia, a survey of research in the United States on the gonococcus and gonococcic infections, now nearing completion, and a world survey of tropical diseases and facilities for studying them, now in process of publication.

Annual Meeting of Dietitians.—At the eighteenth annual meeting of the American Dietetic Association in Cleveland, October 27-31, the program will include addresses by the following physicians:

Dr. William P. Murphy, Boston, Deficiency States—Anemia.
Dr. Wingate Todd, Cleveland, Food for Fitness.
Dr. Henry J. John, Cleveland, Treatment of Diabetic Children.
Dr. Charles H. Eyermann, St. Louis, Allergy and Its Relation to the Dietitian.
Dr. Morris Fishbein, Chicago, Editor of THE JOURNAL, Food Fads and Fallacies.
Dr. Charles C. Higgins, Cleveland, Dietary Management of Urinary Lithiasis.
Dr. Donald J. Barnes, Detroit, Newer Developments in the Treatment of Rickets.
Dr. Victor G. Heiser, New York, World Wide Adventures in Nutrition.

Society News.—Dr. Daniel C. Patterson, Bridgeport, Conn., was elected president of the New England Surgical Society at its annual meeting in Manchester, N. H., September 27-28; Dr. David W. Parker, Manchester, vice president; Dr. John M. Birnie, Springfield, secretary, and Dr. James R. Miller, Hartford, Conn., treasurer. The next meeting will be held in Bridgeport.——Dr. Charles M. Griffith, Washington, D. C., was elected president of the Association of Military Surgeons of the United States at its annual meeting in New York, October 5; Dr. Harold D. Corbusier, Plainfield, N. J., was elected one of the five vice presidents to fill the vacancy left by Dr. Griffith's advancement to the presidency. Among other speakers at the meeting, Dr. Dean Lewis, Baltimore, read a paper on the history of military surgery.

Western States Have Highest Automobile Fatality Rates.—With a 15 per cent increase over 1933, automobile fatalities reached a new high point in 1934, when 33,980 persons were killed in or by motor cars, causing a death rate of 26.9 per hundred thousand of population. In addition, 1,789 persons were killed in collisions between automobiles, railroad trains and street cars. These figures compare with 29,323 deaths in 1933 and a rate of 23.3 per hundred thousand of population. Nevada's automobile death rate of 73.4 was the highest in the nation. Wyoming was second, 45.25; Arizona third, 44.85, and California fourth, 42.62. The bureau of the census directs attention to the fact that the states with the highest rates are in the West rather than the thickly populated Eastern states. While this situation may be brought about by the deaths of transients, there are no statistics at present to prove this point. The lowest rate in the United States (14.6) was for Rhode Island. Only three states, Kansas, Delaware and New Hampshire, showed a decrease in the death rate last year compared with 1933. Gains over 1933 in the remaining states ranged from Connecticut's 2.07 per cent to Montana's 72.13 per cent. With the exception of Vermont the percentage of gain was greatest in the Middle West, Far West and South. New Mexico's gain of 42.12 per cent was the second largest in the nation. Mississippi was third, with an increase of 41.02 per cent. Vermont had a gain of 38.52 per cent, in contrast to New Hampshire's decrease of 23.14 per cent. In Illinois, fatalities increased 17.04 per cent last year compared with 1933. The gain in New York was only 3.49 per cent.

Foreign Letters

LONDON

(From Our Regular Correspondent)

Sept. 28, 1935.

A Survey of Health Services During the King's Reign

The report of the ministry of health for 1935 contains a review of the health services during the twenty-five years of the king's reign. The ministry was established in 1919 to take over the functions of national health insurance and the local government board. National health and pensions insurance is pronounced one of the most memorable developments of public health. In England and Wales 16,450,000 persons are insured, and in the last ten years more than $750,000,000 has been distributed in sickness and maternity benefits and more than $450,000,000 in medical benefits. The provision of pension benefits has steadily expanded and now includes 664,000 old age pensions to persons between the ages 65-70, 657,000 widows' pensions (supplemented by 271,000 additional allowances with respect to children) and 15,000 orphans' pensions.

The expansion of local government services is shown by the fact that the total expenditure has risen from $735,000,000 in 1911 to $2,535,000,000 in the last completed year of the account. The London County Council alone expended $190,000,000, a sum comparable to the budget of some nations. This expenditure on health is defended as a long term investment. The standardized death rate from all causes has fallen from 13.2 per thousand in 1910 to 9.3 in 1934. Some changes in mortality, which may be attributed in part to the operation of public preventive and clinical services, are shown in the accompanying table.

Changes in Mortality

	1910	1933
Infant mortality per 1,000 live births at ages 0-1 years..	105	64
Death rates per million children living at ages 5-15.....	2,491	1,882
Standardized death rates per million from tuberculosis..		9
Respiratory	988	63
Nonrespiratory	434	160
Standardized death rates per million from typhoid.....	53	5

Systematic services for the prevention and treatment of tuberculosis and for maternity and child welfare have come into being, and schemes to aid the blind are now operating all over the country. In 1910 there was no mental deficiency service. In 1913 accommodation was provided for severe cases, the training and employment of defectives, and the treatment of early mental illness. Other advances noted are the provision of postgraduate medical training facilities and the promotion of medical research. Nineteen thirty-four was a record year in two respects: the death rate from all forms of tuberculosis and the infant mortality were the lowest on record. But there was no decline in the maternal mortality rate, and measures are now being taken by the ministry to grapple with this problem. The continued decline in the incidence of smallpox (variola minor) became still more marked in 1934, when the number of cases was fewer than in any year since 1918. For the first time in six years London was free from smallpox on July 12, 1934, so that the smallpox hospital could be closed.

Postgraduate Medical Study in London

The facilities for postgraduate study in London, whether by physicians in general practice or by those who want to cultivate some special branch, are considerable. The twelve undergraduate medical schools are almost entirely devoted to the teaching of students but at times provide a postgraduate course, in some cases confined to their old students and in others open to the profession. At five of the medical schools an intensive

week-end course of lectures for graduates is now in progress and takes place every year at the opening of the medical session in October. In the main, graduate teaching is done at the general hospitals that are not undergraduate medical schools, and at the special hospitals. Most important is the recently established British Postgraduate Medical School, which was opened in May by the king. It contains 388 beds, which are being rapidly increased to 534 by the erection of a new block. The school has no special departments, since it was thought best that graduates who require instruction in the specialties should obtain it at the numerous special hospitals of London. There are four departments—medicine, surgery, obstetrics and gynecology—each under the control of a professor appointed by the University of London, assisted by readers, also appointed by the university, and assistants appointed by the school. It is hoped to include on the staff the leading teachers and authorities available in London and the provinces, and also some from other parts of the world. Refresher courses of an intensive character are provided for physicians in general practice. They last a fortnight and are generally held in the summer months. But the most important part of the teaching is that provided for graduates from all parts of the world. In this a departure has been made. Graduate instruction has usually consisted almost entirely of courses and lectures covering a set syllabus. This will form part of the instruction, but more importance is attached to providing the students with a large amount of clinical material at which they can work under instructors, all of whom have special experience. Carefully prepared clinical lectures are given from time to time. The students also attend regular pathologic and radiologic conferences and discussions between the different departments. The department of pathology not only takes part in the clinical work but provides demonstrations on laboratory procedures. The fees for the ordinary hospital practice, including tuition, are $26 for one month, $52 for three months and $78 for six months. For the fortnight refresher courses the fee is $26.

Unlike the British Postgraduate Medical School, in which almost all the teaching is done in one hospital, the Fellowship of Medicine and Postgraduate Medical Association arranges for teaching in some fifty general and special London hospitals. Regular courses, lasting from one to four weeks, are arranged in general medicine and surgery. Courses in pediatrics, diseases of the chest, heart or nervous system, gynecology, obstetrics, ophthalmology, dermatology, proctology, orthopedics, fractures, psychiatry, urology, physical therapy, venereal diseases, and anesthetics are given at the special hospitals devoted to these specialities. In addition, various special hospitals make arrangements for courses.

The West London Postgraduate College has been established since 1895. The work is carried on at the West London Hospital, which is entirely devoted to postgraduate teaching. Graduates are appointed clinical assistants for three or six months without extra charge. A special clinic for the treatment of venereal diseases (male and female) is held all day. Operations at which graduates are allowed to assist take place daily at 2 p. m. The fees for hospital practice, including all ordinary demonstrations and lectures are $10 for one week, $30 for one month, $45 for two months and $60 for three months. Instruction in the administration of anesthetics is given at the rate of $15 a month.

The North-East London Postgraduate College is attached to the Prince of Wales's General Hospital, which contains 228 beds and is entirely devoted to graduate teaching. Facilities are afforded to graduates for general hospital work or for work in special branches. The fee for general hospital practice is $10 for one month, and $25 for three months. For the special departments the fee is $15 for one month and $30 for three months.

A postgraduate school of radiotherapy was established in 1930. It is held at the Radium Institute and at the Mount Vernon Hospital. The Institute of Medical Psychology holds courses in psychotherapeutic theory and method. A five-week introductory course of twenty lectures, for which the fee is $15, is now in progress. The London School of Hygiene and Tropical Medicine holds a six months course for the Diploma in Tropical Medicine and Hygiene, which lasts from October to June. It consists of instruction in clinical and laboratory work and in tropical hygiene. The fee is $200.

The Treatment of Casualties of the Quetta Earthquake

The appalling disaster of the Quetta earthquake has been reported in a previous letter to THE JOURNAL. As the medical authorities decided that recovery of the bodies buried beneath the débris would have been dangerous, the exact number killed is not known, but it is estimated at 40,000 in the whole countryside involved, a district of 130 by 20 miles. In Quetta itself out of a population of 45,000 between 20,000 and 30,000 were killed. The government took prompt and efficient measures for succoring the survivors. Supplies were sent and refugee camps were established. The treatment of the many cases of serious fracture was excellent. At the Mayo Hospital, Lahore, the organization was of the standard of the highly specialized fracture clinics of Europe or America. All cases of fracture of the spine, pelvis or femur were treated in wards allotted to each kind of fracture. Balkan frames, fitted with adjustable pulleys, weights and traction cable were available for all cases. Several dozens of fractured femurs were treated by stainless steel pins transfixing bone. Cases of fractured pelvis were treated by pin or wire traction applied to both lower limbs. The cases of fractured spine were reduced on a special table and given the best possible chance. Old-fashioned padded splints were conspicuous by their absence. This work was carried out under the directions of Lieut.-Col. V. R. Mirajkar, professor of operative surgery, King Edward Medical College, and superintendent of the Mayo Hospital.

PARIS

(From Our Regular Correspondent)

Sept. 23, 1935.

Changes in Social Insurance Law

In a recent letter attention was called to the many complaints regarding the administration and the deficits in some of the primary disbursement offices of the social insurance organization. To balance its budget, the French government has attempted to make economies in all its departments. Although only in existence since 1930, the social insurance law, which combines sickness and maternity compensation and invalidity and old age pensions, has been modified a number of times. The question of social insurance had been carefully studied for ten years before being made compulsory. The practical application of the principle has revealed many defects. One of the chief differences between the law in France and the laws of other European countries is that the insured sick individual can choose his own medical adviser, who is paid directly by the insured. The latter then presents his statement of expenditures for physicians, dentists, drugs and apparatus to the primary caisse or bureau to which he belongs and is reimbursed to the extent of about 85 per cent. Owing to dissatisfaction on the part of both the insured and the medical profession, a complete revision of the law has been ordered by the secretary of labor, to whose department the execution of the law is assigned. To understand some of the important changes, a brief outline of the present organization will precede the list of changes proposed by M. Froissard as outlined in the September 15 issue of the *Siècle médical.*

The premiums from the insured and those from their employers are forwarded every three months by the latter to

a central office in the capital of each department, there being eighty-six departments in France. The insured and the employer each pay a sum based on the average total earnings of the insured. There are four classes of premiums, varying according to the annual wages of the insured. Every insured person who earns 15,000 francs (about $1,000) and has no children is included in the social insurance law. If there is one dependent child, the limit is higher (20,000 francs) and this rises to 25,000 for those with three or more children. Thus about 60 per cent of all workers are subject to the law.

The departmental receiving office divides its receipts into two parts. One of these is sent to the primary caisses or disbursement centers, which pay sickness, death and maternity claims. Any money left over is sent to another bureau, which up to the present time formed a union with other similar caisses of the corresponding department, to act as a reinsurance company if there should be a deficit in one of its constituent primary caisses. It was the original intention of the law's sponsors that any excess of receipts over expenditures of the primary caisses should be expended for public hygiene purposes, but this has never been done.

The second half of all receipts by the central departmental bureau is sent to primary saving centers, to be paid out for invalidity and old age benefits. Since a large percentage of the insured will not reach the old age limit (60 years) for many years to come, the excess of receipts over expenditures has been placed in a government guaranty fund, to which the state itself has also added 540 million francs (about 36 million dollars), so that as old age pensions become due in future years there may be ample funds to meet the demand. One of the cuts in force since August is to diminish this sum contributed to the guaranty fund (for invalidity and old age pensions) from 540 to 140 million francs. There has been considerable criticism about the manner in which the rapidly accumulating guaranty funds have been invested in unprofitable enterprises. The fact that it will be many years before the majority of those now insured will reach the age of retirement has been one of the defects of the present law.

One of the first reforms proposed by the committee of experts chosen to suggest changes in the law was to cut down the government's contribution to the guaranty fund from 540 to 140 million francs. A reduction in the amount of the annual premiums paid by the insured and the employers was found to be impossible because the diminished receipts would not suffice to pay the various kinds of benefits.

Instead of having so many central receiving offices for each department with a corresponding large number of officials, there will be only one office of this kind for each postal region. This will be a vast saving in administrative costs. An effort to limit the number of primary distributing centers or caisses was unfortunately abandoned, so that many remain each with only a few hundred insured. The unions for reinsurance of the primary caisses or disbursement centers have been discontinued and the money formerly sequestered for these organizations will be devoted to public hygiene work. The reinsurance will in the future be the duty of the central guaranty fund. Thus about 30 per cent of the receipts for sickness, death and maternity benefits can now be utilized in preventive work. This figure (30 per cent) is somewhat chimerical, because many of the primary caisses or disbursement bureaus for sickness, death and maternity will probably have a deficit during the present year and surely will next year.

The discontinued reinsurance bureaus exercised an important control over the expenditures of these primary disbursement offices, because the reinsurance bureau guaranteed the lower bureau's finances. A control will now be lost; hence this modification of the law has been vigorously criticized. Allowance for pharmaceutical specialties, of which there are thousands in France, have been reduced from 85 per cent to 50 per cent. This will be a great saving for the disbursement offices.

Many simplifications of the now cumbersome paper work have been ordered.

Death benefits, instead of being paid by the same office which reimburses sickness and maternity claims, will now be paid by the savings fund.

The unemployed will be considered as regularly insured, provided they have registered for work as soon as the latter can be obtained.

The method of remittances of premiums has consisted in purchasing special social insurance stamps at a postoffice. This will be dispensed with. One needs only to read the list of changes in the present social insurance law to realize that, although it may be of benefit to those whose earnings do not provide for sickness, maternity, death, invalidity and old age, the execution of such a law demands an incalculable amount of paper work ("paperasserie" in French) and a very large staff of officials.

Energetic Antinoise Campaign in Paris

Continuing his energetic efforts to suppress unnecessary noise, the prefect of police has just issued instructions regarding the treatment of the city's inveterate radio sinners. If one's neighbor's radio shrieks at an hour when sleep is, or should be, the chief task of quiet-loving citizens, the police can be informed by telephone and an officer will be sent at once. After investigation, a report will be made and legal proceedings commenced to suppress further radio sinning. Much has already been accomplished here, by restricting the hours during which automobile horns can be used. The chief offender is still the motorcycle, but this question is being studied and soon Paris will seem like a village at night.

First Brucellosis Congress Held in France

The growing importance of Brucella infections has led to the formation here of a society, composed of clinicians, bacteriologists, hygienists and veterinary surgeons. The first meeting of this society was held at Avignon, in the south of France, in June. It is well known now that Malta fever, now commonly called undulant fever or brucellosis, and contagious abortion in cattle are caused by the same etiologic agent, Brucella. In human beings, brucellosis is commonly transmitted through raw cow's milk or unpasteurized dairy products. Among the latter are to be especially mentioned, in France, cheese made from the milk of goats.

In addition to members of the French medical and veterinary professions, there was a relatively large number of Italian and English visitors.

The first day was devoted to the clinical aspects of undulant fever. Cannavo of Libya reported his own case of this form of infection, characterized by the appearance, on the fiftieth day, of a choroiditis. Chantegreil added three cases all verified by a positive seroreaction, complicated by a phlebitis in one case. Meerseman observed an acute encephalitis confirmed at necropsy, the inflammatory lesions being chiefly situated in the subcortical tissues.

Bruschettini of Genoa, Italy, has found that an egg medium is the simplest to employ in the bacteriologic diagnosis. He had been able to confirm the existence of two types of Brucella infection. Barbary and Faure-Brac of Nice strongly advocated the more widespread use of the intradermal reaction. Undulant fever is the most serious disease that threatens the raising of cattle, sheep and hogs. Every prophylactic measure must be employed, in the form of the intradermal reaction, to prevent spread of the infection to human beings and among animals.

Guigni of Bologna, Italy, was enthusiastic about the use of a specific vaccine containing from 5 to 10 million bacteria per injection, which is followed in the majority of cases by a rapid regression and cure of human brucellosis. Picard of Arles (France) was not so enthusiastic, as he had had only a large number of failures.

Cambessedes of Paris believed that anaphylactic shock reaction in the form of vaccinotherapy and chemotherapy were strongly indicated in the treatment of human brucellosis. A number of other speakers were of the opinion that such a form of treatment is not to be recommended, because it is apt to cause exacerbations of a most serious character.

Dubois and Sellier of France have used as a prophylactic measure a polyvalent antibrucellosis vaccine during the past five years in 5,000 people exposed to animal infection. This form of prophylaxis ought to be used by every butcher, veterinarian and cattle raiser.

Jullien of France warmly advocated centers like the one at Joyeuse, in which those suffering from undulant fever can be isolated. Many of these patients live in mountainous regions, are difficult to get to and thus cannot be given the daily observation, which is needed.

Every small community should be provided with a diagnostic and prophylactic center. These stations would have an important educational effect on the rural population in showing how the disease is recognized and treated. The economic loss in regions where undulant fever exists is enormous. If the infected individuals can be returned to work in twenty-five days, as is now possible at the Joyeuse center, the money spent in such a direction will be amply repaid.

Louis Roux, a bacteriologist of Lausanne, called attention to the association of brucellosis and tuberculosis. In seven mammary cases this combination was present. The Bang bacillus seems to be the primary invader.

Finzi of Italy does not believe that dead vaccines are of any value and uses only living ones. Preventive vaccination of cattle is the only way of checking the spread of brucellosis.

Hayat of Tunis found that in laboratory animals infected with brucellosis a superadded tularemia might occur. He had observed agglutination reactions toward both infections in the blood of such animals.

Jullien of Paris stated that in cases of prolonged fever one ought always to take into account the possible presence of kala-azar, which is far more widespread in southeastern France and Mediterranean countries than is brucellosis.

In resolutions passed at the close of the congress, the establishment of an international bureau for the study of human and animal brucellosis was decided on.

The Birth Rate and Mortality in Some European Countries

According to Moine's statistics reported at the July 23 meeting of the Academy of Medicine, the decreased ratio of births to deaths dates back as far as to the decade 1811-1820, when there were only 318 births to 10,000 inhabitants. This figure gradually decreased to 281 from 1851 to 1870, during which most other European countries registered an increase in their natality, which continued to the end of the nineteenth century. From 1871 to 1880 there were only 254 births to 10,000 inhabitants, as compared to 308 in Switzerland, 323 in Belgium, 354 in England, 362 in Spain, 369 in Italy and 391 in Germany. Since 1880 there has been a decrease in the number of births in all these countries, so that in 1933 the last places in the list were occupied by England (144) and Germany (147). Only Spain and Italy showed a relative increase to 262 and 232 respectively. The number of births since 1880 has decreased by 23.5 per cent in Spain, 35.8 per cent in France and Italy, 46.8 per cent in Switzerland, 48.9 per cent in Belgium, 59.3 per cent in England and 62.5 per cent in Germany. The coefficients of fecundity for each 10,000 women between the ages of 15 and 49 years was 868 in 1901 for France, 1,040 for England and 1,440 for Germany. These figures show a marked decline in 1933. The coefficients of fecundity in 1933 as compared to those of 1901 show a decrease of 64.6 per cent for Germany, 50.2 per cent for England, 44.2 per cent for Switzerland, 35.5 per cent for Italy and only 29.1 per cent for France. This improvement in France is the result of the work of various organizations that have encouraged the raising of larger families. As to the number of deaths for each 10,000 inhabitants in 1933, the percentage for France was 158, Germany 112, Italy 137, England 123, Spain 164, Belgium 134 and Switzerland 114. If one considers the mortality of each country according to the population distributed according to the type (city and country), France between 1920 and 1922 had only 149 deaths, against 165 in Germany, 226 in Spain and 156 in Italy.

If the age of those who have died is taken into consideration, one finds a marked decrease in the mortality at certain ages since 1900 in France. This diminution of mortality has been in part due to a smaller number of deaths from tuberculosis. In 1880 there were 500 deaths from this disease per hundred thousand inhabitants in Paris. At the present time there are only 176, a decrease of 66 per cent as compared to that of the general mortality of 48 per cent. As a result of the fight against venereal diseases in France, more than 20,000 new-born infants are saved annually.

Two problems the solution of which will be aimed at in the future are, first, to increase the natality by decreasing the mortality, and, secondly, to struggle against infant mortality.

BERLIN

(From Our Regular Correspondent)

Sept. 2, 1935.

Treatment and Prognosis of Severe Gastric Hemorrhages

The "Treatment and Prognosis of Severe Gastric Hemorrhages" was discussed at a recent session of the Berlin Medical Society. Professor Umber emphasized that few clinical reports with detailed statistics have been published. This accounts for the discrepancy between the German opinion, supported by Morawitz, Bergmann and others, to the effect that death from a severe bleeding induced by an ulcer is rare, and the English statistics that give a mortality of from 18 to 23 per cent. Umber therefore had statistics prepared on the cases occurring in his clinic during the past fifteen years, which disclose that, in 1,852 gastric and duodenal ulcers, 433 severe manifest hemorrhages occurred, associated with vomiting of blood and tarry stools. In forty-one cases death occurred as a result of excessive hemorrhages. Thus it appears that the mortality from hemorrhages was 9.5 per cent and the mortality from hemorrhages associated with ulcers was 2.2 per cent. Hence Umber considers the mortality from severe hemorrhages due to ulcer as much higher than is commonly supposed. A large percentage of the fatal cases occurring in his clinic presented severe hemorrhages due to erosion of the walls of the gastroduodenal artery, the gastrica sinistra or the gastrica dextra. If the hemoglobin drops to from 20 to 30 per cent, serious danger for the patient arises. From the standpoint of treatment, rest and narcotics are indicated; also hemostatic, hypertonic solutions of sodium or calcium salts. If the stomach is full of blood, and a tendency to vomit arises, gastric lavages with ice-water are advisable, since they not only give the patient subjective relief but have also a hemostatic effect. Nutrition should be ample; for long continued undernutrition furthers the hemorrhagic tendency. For this purpose iced butter pellets are recommended; in addition, doses of vitamin C twice daily. Rectal clysmas of high calory content are, however, rejected, as they increase the secretion of

gastric juice. In pronounced anemia, blood transfusions are advisable, sometimes repeated on several successive days. No more than 500 cc. should be injected, since, as a result of an increase in pressure, thrombi may easily develop. A blood transfusion should be compulsory before every operation. When the hemorrhage has been checked, iron preparations should be given to combat the anemia. Of 433 cases of gastric hemorrhage, an operation was performed in thirty-nine, resulting in seven fatalities; in three of these patients it was found later that a carcinoma had been present. As the source of hemorrhage, Umber found much more frequently a gastric ulcer than a duodenal ulcer, which agrees with the statements of others. Severe hemorrhages due to ulcer should be treated first internally; in recurrences and in ulcer hemorrhages that show no tendency to thrombose (suspicion of arterial hemorrhage) an operation is indicated.

From the surgical point of view, Professor Reschke likewise found hemorrhages due to ulcer more frequent than is generally supposed. A hemorrhage may be regarded as grave when it is doubtful whether a patient can be saved by internal treatment. Reschke had good success with surgery and only a slight mortality from pneumonia and peritonitis. The mortality from hemorrhage due to ulcer is higher in men than in women. The normal method of operation is resection. If a blood transfusion is allowed to precede an operation, the intervention is not dangerous. It is his observation, however, that blood transfusion often does not check the hemorrhage but, on the contrary, makes the next hemorrhage worse. Danger of a recurrence of a hemorrhage is especially great two days after the transfusion. The operation should not be undertaken until the patient has recovered his strength as the result of a large transfusion of from 1,000 to 1,500 cc., together with a like amount of solution of dextrose. If the patient does not recover his strength, the transfusion should be allowed to suffice. Early operation during the first twenty-four to forty-eight hours, as proposed by Finsterer, does not appear necessary.

In the discussion, Professor von Bergmann emphasized the difficulty of distinguishing between mild and severe hemorrhage. He is opposed to morphine because it retards the gastric motility and through collection of blood in the stomach leads to vomiting; it also increases nausea. He has not observed that the tendency to hemorrhage is accentuated by hunger. The fewer remedies that are given to promote circulation in connection with hemorrhage, the easier it is to check the flow of blood. Since the introduction of the preoperative blood transfusion, von Bergmann is no longer inclined to oppose an operation. Professor Kalk presented statistics of his hospital department in Berlin. It appears that, in ten years, of 157 severe hemorrhages, thirteen, or 8.3 per cent, ended fatally; but only in six of the thirteen fatal cases was hemorrhage the sole cause.

The Physiology of Work

Professor Basler, an authority on the physiology of work, who has been appointed to a chair at the University of Breslau, spoke recently on the "Tasks of the Physiology of Work." As the two chief tasks of the physiology of work he designated (1) the determination of the most favorable, least fatiguing work method, and (2) the discovery of the persons who are best adapted for a certain form of activity. To perform these tasks adequately, and particularly to measure the amount of effort necessary to do a certain piece of work, requires more than ordinary care in research. The most universal type of work is walking. The principal movements are those of the sole of the foot and of the hip. The movements in the intermediate joints needed to round out the principal movements are of secondary importance. In walking, the lifting of the body about 4 cm. brings about a frictional resistance of the joints. The energy consumption, measured by respiratory metabolism, varies with the number and length of the steps. The optimum is 35 calories (body weight 80 Kg.) with the number of steps at 87.5 and the length of step 58.7 cm. The most practical load under the most favorable conditions is 43 per cent of the body weight. The most favorable load is that of the back, whereas the exertion, without corresponding increase of the energy consumption, is greater, the greater, in any activity, the static component becomes (for example, carrying a weight in the hand). The work to be performed may become thereby absolutely impossible. There is as yet no perfectly reliable method for the objective measurement of fatigue. The unconscious shortening of the length of step after a long march is of considerable value as a criterion of fatigue. This is determined by counting the number of steps taken in a specified stretch. The second method is based on the "decrease in the physiologic height" of a person. The examinee is allowed to walk past a board on which are pasted a number of horizontal strips 1 cm. wide. The examiner with a field-glass notes the strip that is in line with the crown of the head of the examinee at the highest point while the examinee is walking.

With regard to utensils, for example, the wheelbarrow commonly used in Europe is much less practicable than the Chinese wheelbarrow, in which the center of gravity of the load lies directly over the axis of the wheel.

Basler called attention to a certain test of fitness for a given type of labor which he considered important; namely, a test of the ability to stand on one's feet for long periods of time. To determine quickly the resistance of the vascular apparatus of the legs of an examinee, the examiner measures, instead of the increase in volume, the increase in the girth of the lower leg, using for the purpose a flexible tape, which is fastened about the leg and which a special device keeps at a uniform tautness. The girth of the leg can be read from a scale. If the leg of an examinee increases unduly in size after he stands for one hour, he is advised to avoid those occupations which require a workman to stand for long periods.

AUSTRALIA

(From Our Regular Correspondent)

Sept. 3, 1935.

Sixth Australian Cancer Conference

The influence of the fourth International Congress of Radiology held at Zurich in July 1934 was felt at the sixth Australian Cancer Conference at Canberra, May 14-17. Another feature of the conference was the more active part played by the Royal Australasian College of Surgeons. This is a significant development, as there was a danger that the element of bureaucracy was playing too large a part in the proceedings. Cancer of the breast was discussed from the surgical and radiotherapeutic aspects but no finality was achieved owing to difficulties of comparing the statistics.

RECOMMENDATIONS REGARDING STANDARDIZATION

1. It is desirable to introduce in Australia, wherever it is not already in use, the international method of measuring x-rays in terms of the roentgen unit, as prescribed by the International Congress of 1934.

2. There should be provision for the testing of x-ray equipment in all the characteristics essential for its effective operation.

3. There should be in Australia a single primary standard for the measurement of the international roentgen unit.

4. There is urgent need in Australia for facilities for the testing, examination and calibration of scientific apparatus and instruments used in radiotherapy, and for the carrying out of scientific investigations connected with: (*a*) the purely physi-

cal aspects of radiotherapy; (*b*) the biophysical aspects of radiotherapy, and (*c*) the performance of x-ray equipment.

5. Such facilities should exist in several centers and in particular in the two largest cities—Sydney and Melbourne—where provision should be made for the continuance and extension of existing physical services.

6. In order to secure the best results, provision should be made for close coordination and for the dissemination of relevant information.

7. The federal government should subsidize these activities financially.

8. The work should be coordinated and controlled by a special board, the constitution of which should be arranged by the Commonwealth Department of Public Health and the Council of Scientific and Industrial Research in consultation.

Professor Laby of Melbourne in support of the recommendations drew attention to the remarkable developments in the physics of cancer treatment that had taken place abroad. In England, France, America, Germany, Sweden and other countries a number of physical research laboratories to serve cancer clinics have been set up. These laboratories carry out roentgen and radium measurements and investigate the use of x-rays of higher voltage than those hitherto used and the possibilities of artificially produced radioactivity.

He stressed the necessity of obtaining some degree of accuracy in the measurement of x-rays and gamma rays from radium used therapeutically and to introduce into Australia the international method of measuring x-rays in the terms of the roentgen unit. This involves (*a*) the measurement of the quantity (dosage) of x-rays by air ionization methods, (*b*) the determination of their quality according to rule 5 of the International Recommendations, and (*c*) measurement of radium, radon and gamma rays.

He drew attention to the fact that precision of physical measurements cannot be made by methods that can be specified for all time, and they must be in the control of those who are engaged in original research in the branch of physics to which the standards relate, and who are leaders in that branch of physical research. This principle is fully accepted in the national physical laboratories referred to. If Australia is not to be behind these countries, it will need to do the same.

Rule 5 of the international recommendations states: "The specification of dosage shall include a statement of the quality of the radiation. For exact physical measurements the quality of the radiation may be specified in terms of the complete absorption curves in copper or aluminum. For most practical purposes the quality may be expressed by the first and second half-value layers of the same materials."

Activities of Commonwealth Radium Laboratory

The increase in the use of radon can be gaged by the quantities issued by the laboratory at Melbourne (table 1).

Ninety-two per cent of the radon issued during 1934 was mounted in the form of needles. Of the remainder, 7 per cent was issued in tubes for gynecologic use and 1 per cent in small gold implants. Of the radon issued in needles, 66 per cent was issued in needles of 0.8 mm. platinum filter, 25 per cent in needles of 0.5 mm. platinum filter and 1 per cent in needles of 0.4 mm. platinum filter. The tendency toward the use of the more heavily filtered needles is evident from these figures. At the Melbourne Hospital and St. Vincent's Hospital radon needles of 0.8 mm. platinum filter are used exclusively.

Protection arrangements have been intensified by the following:

(*a*) All lead screens behind which radium or radon is handled are to be increased in thickness to 4 inches of lead.

(*b*) Experiments are being carried out with a semiautomatic radon purification plant, which, if found satisfactory, will replace the existing plant. Such a semiautomatic plant would greatly reduce the exposure received by the operator, and, if the plant can be made sufficiently compact, it could be surrounded by several inches of lead.

(*c*) A device is being installed whereby any leakage of radon into the air of the laboratory will automatically cause the ventilating fan to be set in operation. Such a device should assist in keeping the laboratory free from radioactive contamination.

TABLE 1.—*Increase in Use of Radon*

	Millicuries
1929 (six months)	3,720
1930	11,830
1931	17,030
1932	14,460
1933	18,390
1934	23,210

Pernicious Anemia in Australia

It is interesting to note in the Australian Year Book the death rate from pernicious anemia and the effect thereon of the introduction of liver therapy.

Prior to 1928 deaths from pernicious anemia were not recorded separately in the Australian Year Book. Consequently, prior to that date the total includes other anemias as well as chlorosis.

In that year Minot and Murphy announced their discovery. It is interesting also to note the dates on which the work of Minot and Murphy appeared. Preliminary observations had been published in THE JOURNAL (Aug. 14, 1926, p. 470; Sept. 3, 1927, p. 759) but the work on the reticulocyte response was published in 1928 in the *American Journal of the Medical Sciences* (**175**:581 [May] 1928). That the death rate should have been almost halved the same year is an indication of the ready acceptance of the new treatment by Australian physicians.

One effect noted in Australia is the quickening of interest in the anemias generally, their more complete investigation and their more thorough classification for treatment purposes. It is possible that this has accounted for the apparent rise in the figures for the late years shown in table 2.

TABLE 2.—*Yearly Deaths from Pernicious Anemia*

Year	Males	Females	Total	Other Anemias and Chlorosis
1920	161	192	353	—
1921	158	212	370	—
1922	192	166	358	—
1923	244	246	490	—
1924	186	232	418	—
1925	208	225	433	—
1926	220	230	450	—
1927	151	240	391	—
1928	102	116	218	43
1929	119	119	238	—
1930	94	141	235	50
1931	121	159	280	52
1932	133	155	288	30

International Union Against Cancer

Correspondence had passed between the commonwealth government and the international union in connection with the suggested adhesion of Australia to it. The commonwealth government had decided to defer any action regarding adhesion to the International Union or the nomination of official representatives on the council of direction until the matter had been discussed by the Australian Cancer Conference, and the president of the international union had been informed accordingly.

The conference, after discussing the matter, expressed the opinion that the question of joining the International Union Against Cancer was a matter for the cancer organization in each state to decide on individually, and that it be left in the hands of each state organization to take such action as it considered desirable in this respect.

RIO DE JANEIRO

(From Our Regular Correspondent)

Sept. 15, 1935.

The First Reported Case of Undulant Fever

Dr. José Guilherme Lacorte, head of the laboratories of the Instituto Oswaldo Cruz, in a lecture before the Academia Nacional de Medicina of Rio de Janeiro reported bacteriologic and serologic studies in a case of undulant fever, the first one seen in Rio de Janeiro. The bacteria developed on a solid culture medium placed in an atmosphere overcharged with carbon dioxide. The medium was prepared according to Huddleson's technic. The bacteria were not motile in broth cultures of twenty-four or forty-eight hours examined by the hanging drop method, except for exaggerated brownian movement. They are rod shaped with round ends varying between 0.2 and 0.4 micron in width and 0.5 and 1 micron in length and are gram negative. Colonies that develop in petri dishes containing ordinary gelose after forty-eight hours resemble pin heads between 0.2 and 0.4 mm. in diameter; they are convex, transparent, humid, shining and smooth, with complete and regular contours and a center darker than the rest of the colony. As they grow older they turn brown and their borders become irregular. The bacteria, when placed in simple broth, produce slight turbidity after twenty-four hours and intense turbidity and precipitation after forty-eight hours; in gelatin at 20 C. they produce moderate proliferation without liquefaction; on potato medium they grow poorly and turn the medium dark; they fail to form indole and to reduce nitrates to nitrites, and they do not form acid or gas. The organism proliferates well in Huddleson medium, when it contains fuchsin in the proportion of 1 to 25,000, thionine in the proportion of 1 to 30,000 or methyl violet in the proportion of 1 to 100,000. The speaker concluded that the organism was Brucella melitensis; however, the patient was a hog slaughterer, a fact that would make more plausible, his infection with Brucella suis. The fact that the bacteria grew in a medium placed in an atmosphere overcharged with carbon dioxide, which is a characteristic of Brucella abortus, is also surprising. However, the bacteria isolated by the speaker behave as Brucella melitensis with respect to the formation of hydrogen sulphide and in not taking certain stains.

Local Insulin Therapy in Pleural Thickening

Dr. Santos Fortes reports satisfactory results with local insulin therapy in about 100 cases of pleural thickening. The treatment consists of subcutaneous injections of insulin in doses varying from 0.2 to 1 cc., associated with the administration of sugar, either in water or in candies. The injections are made at the level of the pleural lesions and are given every other day in a number varying from twelve to ninety. Satisfactory results were obtained in thirty of a group of thirty-four cases. The treatment was discontinued in the remaining four because of hypersensitivity to insulin. Hypoglycemia, which appeared in some cases during the course of the treatment, was controlled by the administration of sugar. The treatment gives the best results in postpneumonic pleural thickening of the type described by Morquio and also in some cases similar to those described by Bacmeister as chronic pneumonia, the prognosis of which has been considered as fatal. The speaker, with Argerich, believes that the local action of insulin therapy is probably due to a trophic action of insulin on the tissues by which glycopexis in the tissues is promoted.

JAPAN

(From Our Regular Correspondent)

Aug. 29, 1935.

Vital Statistics

In its recent report, the statistical bureau of the cabinet states that according to the census of 1930 the death rate of the nation was less than 20 per thousand of population. The death rate of infants was highest, having been 140 for boys and 124 for girls per thousand babies. When boys grow to be 16 or 17 years old and girls 17 or 18 years old, only three fourths of them have survived their contemporaries. Just half the number of men of 54 or 55 and women of 55 or 56 have died, and one fourth of the men of 69 or 70 and women of 73 or 74 survive. The average span of life is roughly estimated to be 50 years, but the average span of life of babies under 1 year old was 44.82 years in boys and 46.54 years in girls. When they were 3 years old their life expectancy was the longest; that is, 52.54 years for boys and 53.59 years for girls. As the age advances the expectancy gradually grows less, and at the age of 20 men had 40.18 years and women 42.12 years more to live. When men were 32 years old and women were 33 or 34 the figures showing their ages and their life expectancy was just the same. At the age of 50 men had 18.49 years more to live, and women 21.67 years.

The death rate by age groups is shown in the following table:

Ages	Male	Female
0	140.1	124.1
1	43.1	42.1
2	22.4	22.7
5	6.4	7.1
10	2.6	3.0
15	5.0	7.3
18	9.1	10.1
20	9.8	10.6
23	9.3	10.2
24	9.0	9.9
30	7.4	8.9
40	9.6	10.1
50	17.5	12.6
60	36.7	24.2
70	80.4	57.7
80	170.2	138.5
90	341.4	322.7
100	604.8	624.3

Junior Nurses

The government believes that expenses incurred by patients should be lessened. Patients now generally have to pay the nurses almost as much as the hospital charges and sometimes even more. Sometimes an expert nurse is not required and a cheaper nurse will do. The home office sees much demand for a practical nurse and will train "junior nurses," who will be able to attend the sick in place of the licensed nurses for a much smaller fee. Junior nurses will be trained for three months in the government institute, while the licensed nurses will be required to take a course of training for two years.

Nearsightedness in Japanese Students

The results of the annual physical examination at the various schools for 1933 has been a matter of great surprise. In the middle grade schools, attended by boys and girls from 12 to 18 years of age, the percentage of the nearsighted has increased in the last twenty years to 36.35 from 15.97 in boys and to 34.56 from 10.40 in girls. In the vocational schools the increase shows almost the same percentage. In colleges and universities about 35,000 students are nearsighted; that is, fifty students out of a hundred. The cause of this increase has been investigated by various medical and educational bodies, such as the school physicians' society, the health bureau of the educational office and the Japan Ophthalmologic Association. They say the cause is, in part, the small type used in dictionaries and textbooks, which children are obliged to read to prepare for

the entrance examinations to the various schools. They are burdened daily by the keen competitive examinations after they reach the age of 10 because of the surplus population all over the country. Secondly, modern students have weaker constitutions than the previous generations. Thirdly, the old fashioned buildings are poorly lighted. School buildings now have improved lighting, but the dwelling houses often are far from being well lighted. A sensation was thus created in Tokyo and the authorities have set about to prevent this condition in future generations. On the other hand, some are of the opinion that the children's eyes are weakened in infancy because most of the Japanese children are carried on the backs of maid servants, often facing the strong rays of the sun. With such a vital problem, a general movement for prevention is expected to be undertaken soon by the nation as a whole.

Medical Economics

In the prefecture of Shizouka, with a population of about 1,700,000, reports were made Oct. 1, 1930, by medical practitioners on various topics. According to this report about twenty-nine patients visit a practitioner daily in cities and about thirty-four patients in rural districts, giving an average of 30.5 patients. This includes the old and the new cases at the ratio of 6.9 old cases to 1 new case. Therefore, on an average, about four new patients come to a practitioner each day. The number of the practitioners in this prefecture is 1,013 in all, including 167 general practitioners, 536 physicians, ninety-eight surgeons and sixty-one obstetricians. Of these patients 8.8 per cent could not afford to pay a fee. Out of every thousand patients there were 132 between 15 and 19 years old, and they comprised the largest number.

Marriages

WILLIAM E. DAWSON, Hookerton, N. C., to Miss Winnie Davis Reel of Morehead City, at Kinston, August 25.

WILLIAM MORGAN CHEW, Waynesboro, Va., to Miss Katharine L. Salmon of New York, August 14.

EMILE E. THERRIEN, West Vancouver, B. C., to Miss Bernice McCartney of Langley Prairie, July 24.

HERMAN HARRISON BRAXTON, Chase City, Va., to Miss Anne Norfolk Grimm of Baltimore, June 22.

FREDERICK SIDNEY HOBBS, Vancouver, B. C., to Miss Catherine Bridgman of Calgary, Alta., July 13.

EDWIN J. DEALY, Valhalla, N. Y., to Miss Esther Elizabeth Hinman at New Castle, August 26.

ADAM TYREE FINCH JR. to Mrs. Anne Reynolds Geoghegan, both of Chase City, Va., August 5.

EVERARD T. W. NASH, Vancouver, B. C., to Miss Truda Harvey of Mission City, recently.

ROLAND SMITH CLINTON, Gastonia, N. C., to Mrs. Gertrude Kohn Keller of Charlotte, July 12.

HARRY CARTER DUNSTONE, Ypsilanti, Mich., to Miss Ruth Vogel of Chelsea, August 19.

JOHN B. COUSAR, Bishopville, S. C., to Miss Helen McMillan of McRae, Ga., June 12.

CRAIG C. WALES, Fairfield, Ohio, to Miss Glendora Barnes of Westerville, June 20.

PAUL E. PIFER, Kenosha, Wis., to Miss Phyllis Marie McCue of Cleveland, June 29.

RICHARD J. DIETZ to Miss Evelyn A. Tobin, both of Milwaukee, July 27.

ARCHIE D. EREHART to Miss Mary Herzog, both of Anderson, Ind., July 3.

RALPH B. DAVIS, McComb, Miss., to Miss Gileroy Porter of Ripley, July 17.

JOHN A. ENRIGHT to Miss Cecilia Wocasek, both of Milwaukee, July 6.

SAMUEL SOLOMON to Miss Estelle Suffin, both of Brooklyn, August 16.

Deaths

August Caille ✠ New York; Julius-Maximilians-Universität Medizinische Fakultät, Würzburg, Bavaria, Germany, 1877; College of Physicians and Surgeons, Medical Department of Columbia College, New York, 1881; emeritus professor of pediatrics, New York Post-Graduate Medical School of Columbia University; fellow of the American College of Physicians; past president of the American Pediatric Society; delegate to the International Medical Congress in Berlin; aged 81; at various times on the staffs of the Convalescent Home for Babies, Sea Cliff, N. Y., New York Post Graduate Hospital, Isabella Home and Hospital and the Lenox Hill Hospital, where he died, October 10, of cerebral hemorrhage.

Simeon Edward Josephi ✠ Portland, Ore.; University of California Medical Department, 1877; dean emeritus, emeritus professor of nervous and mental diseases, and one of the founders of the University of Oregon Medical School; past president of the Portland Medical Society and the Portland Academy of Medicine; at one time member of the state legislature; medical superintendent of the Oregon State Hospital for Insane, 1881-1883, and the Oregon State Hospital, Salem, 1886-1887; for many years on the staff of the Good Samaritan Hospital; aged 85; died, August 23.

Alvin Walter Klein ✠ Stockbridge, Mass.; Cincinnati College of Medicine and Surgery, 1889; member of the American Psychiatric Association; served during the World War; physician to the Austen Riggs Foundation; at one time chairman of the board of health of Greenwich, Conn., and on the staff of the Greenwich (Conn.) Hospital; aged 67; died suddenly, September 27, of coronary occlusion.

Willis Newton Smith, Phoenixville, Pa.; Western Pennsylvania Medical College, Pittsburgh, 1892; member of the Medical Society of the State of Pennsylvania; past president of the Chester County Medical Society; on the staff of the Phoenixville Hospital; aged 77; died, September 6, in the Chester County Hospital, West Chester, of nephritis.

Peter John Zeglio ✠ Plainfield, N. J.; College of Physicians and Surgeons, Medical Department of Columbia College, New York, 1882; fellow of the American College of Surgeons; on the staffs of the Muhlenberg Hospital, Plainfield, and the Somerset Hospital, Somerville; aged 75; died, August 3, of chronic myocarditis and arteriosclerosis.

Neil McLean ✠ Devils Lake, N. D.; Minneapolis College of Physicians and Surgeons, medical department of Hamline University, 1906; past president of the Devils Lake District Medical Society; served during the World War; aged 57; died, September 3, in a hospital at Livingston, Mont., of pneumonia and cerebral hemorrhage.

Walter J. Leach ✠ New Albany, Ind.; Louisville (Ky.) Medical College, 1897; president of the Indiana State Medical Association and formerly councilor of the Third District; past president and secretary of the Floyd County Medical Society; on the staff of St. Edward's Hospital; aged 73; died, September 29, of heart disease.

John E. Nevill, Bonham, Texas; Memphis (Tenn.) Hospital Medical College, 1900; member of the State Medical Association of Texas; past president of the North Texas District Medical Society and the Fannin County Medical Society; aged 66; died, August 31, in a sanatorium in Paris, of amyotrophic lateral sclerosis.

James Donald McKinnon, Port Chester, N. Y.; McGill University Faculty of Medicine, Montreal, Que., Canada, 1924; at one time instructor and professor of pathology at the University of Wisconsin Medical School, Madison; aged 36; died, September 5, at the United Hospital, of an accidental overdose of paraldehyde.

Henry Herbert Healy, Grand Forks, N. D.; Rush Medical College, Chicago, 1892; member of the North Dakota State Medical Association; fellow of the American College of Surgeons; surgeon to St. Michael's Hospital and the Deaconess Hospital; aged 65; died, September 1, of carcinoma of the prostate.

Franklin Elijah Spear ✠ Woodsville, N. H.; University of Vermont College of Medicine, 1903; past president of the Grafton County Medical Society; served during the World War; aged 62; on the staff of the Cottage Hospital, where he died, September 5, of acute hemorrhagic pancreatitis.

John B. Kouwenhoven ⊕ Yonkers, N. Y.; College of Physicians and Surgeons, Medical Department of Columbia College, New York, 1895; at one time member of the city health department; for many years on the staff of St. John's Riverside Hospital; aged 64; died, August 27, at Dorset, Vt.

John S. Jackson ⊕ Alpena, Mich.; Detroit College of Medicine, 1905; also a dentist; past president and secretary of the Alpena County Medical Society; aged 63; on the staff of the Donald McRae Hospital, where he died, August 24, of diabetic acidosis complicated by a carbuncle on the neck.

David T. Smith ⊕ Omer, Mich.; Detroit College of Medicine, 1903; past president of the Bay County Medical Society; formerly mayor of Omer; medical director and owner of a hospital bearing his name; aged 54; died, September 8, of coronary thrombosis and cerebral embolus.

Alexander Bell, Toronto, Ont., Canada; Queen's University Faculty of Medicine, Kingston, 1865; medical health officer of the city of Peterborough and county coroner for many years; Civil War veteran; at one time clerk and chief municipal officer of Lakefield; aged 91; died, September 1.

Joseph Anthony Stockler ⊕ Reading, Pa.; University of Pennsylvania School of Medicine, Philadelphia, 1909; member of the American Academy of Ophthalmology and Oto-Laryngology; aged 51; on the staff of St. Joseph's Hospital, where he died, September 9, of heart disease.

Monroe Clayton Sapp ⊕ Cameron, Texas; Tulane University of Louisiana Medical Department, New Orleans, 1907; past president of the Milam County Medical Society; on the staff of the Cameron Hospital; aged 70; died suddenly, August 5, of heart disease.

Vergil Alvin Ross ⊕ Champaign, Ill.; Rush Medical College, Chicago, 1917; served during the World War; assistant health officer and senior medical adviser to men at the University of Illinois; aged 42; was found dead in bed, August 26, of coronary thrombosis.

Frederick Burke Carron, Brockville, Ont., Canada; McGill University Faculty of Medicine, Montreal, Que., 1896; M.R.C.S., England and L.R.C.P., London, 1898; served with the Canadian Army during the World War; aged 64; died, August 25.

Nathan Hunt, National City, Calif.; State University of Iowa College of Medicine, Iowa City, 1872; Civil War veteran; formerly health officer of San Diego County; aged 88; died, August 23, at Sawtelle, of arteriosclerosis and thrombosis.

John Rudd Mahone, Shafter, Texas; Tulane University of Louisiana Medical Department, New Orleans, 1905; at one time member of the state board of health, health officer of Hidalgo County and Cameron County; aged 57; died in August.

Dickson Gillespie Thompson, Waxahachie, Texas; University of Louisville (Ky.) Medical Department, 1877; member of the State Medical Association of Texas; aged 78; died, August 29, of carcinoma of the gastro-intestinal tract.

Edward Augustus Willis, New York; University of Michigan Department of Medicine and Surgery, Ann Arbor, 1902; served during the World War; aged 61; died, September 25, in the Mount Sinai Hospital, of cirrhosis of the liver.

Oscar O'Bannon Martin ⊕ San Antonio, Texas; University of Texas School of Medicine, Galveston, 1930; aged 31; died, August 26, at the Station Hospital, Fort Sam Houston, of injuries received in an automobile accident.

Caspar William Letzerich, Harlingen, Texas; Tulane University of Louisiana Medical Department, New Orleans, 1899; member of the State Medical Association of Texas; aged 60; died, September 1, of coronary occlusion.

William Lewis Peck, Coalwood, W. Va.; University of Louisville (Ky.) Medical Department, 1912; member of the West Virginia State Medical Association; aged 61; died suddenly, August 30, of myocarditis.

Murray Emmet Swinehart, Columbus, Ohio; Starling Medical College, Columbus, 1906; member of the Ohio State Medical Association; aged 59; died, September 9, of idiopathic hypoplasia of the bone marrow.

Henry Johns Owens, Hazleton, Pa.; Jefferson Medical College of Philadelphia, 1896; member of the Medical Society of the State of Pennsylvania; aged 63; died, August 29, in a local hospital, of pneumonia.

Hermann Mond ⊕ New York; Medizinische Fakultät der Universität Wien, Austria, 1920; for many years on the staff of the Mount Sinai Hospital; aged 43; died suddenly, September 17, of coronary thrombosis.

Edward Preston Hale, Lenox, Mass.; Bellevue Hospital Medical College, New York, 1881; member of the Massachusetts Medical Society; bank president; aged 75; died, September 9, of heart disease.

Oliver Edmund Snodgrass, Philadelphia; University of Pennsylvania Department of Medicine, Philadelphia, 1886; aged 71; died, August 27, in Ocean City, N. J., of chronic nephritis and arteriosclerosis.

Minnie James Cantelo Nicholson, Haverhill, Mass.; College of Physicians and Surgeons, Boston, 1915; member of the Massachusetts Medical Society; aged 59; died, August 31, in the Gale Hospital.

Claire Ella Armstrong Russ, Cincinnati; Laura Memorial Woman's Medical College, Cincinnati, 1896; aged 69; died, September 5, of lymphoblastoma with exfoliative dermatitis and toxic nephritis.

Thomas Dana Jacobs, Grandmound, Iowa; State University of Iowa College of Medicine, Iowa City, 1913; served during the World War; aged 54; died, September 2, of coronary thrombosis.

Ned J. Malloy, Gagetown, Mich.; Marquette University School of Medicine, Milwaukee, 1914; member of the Michigan State Medical Society; aged 51; died, August 30, of coronary thrombosis.

Herbert Arthur Wildman ⊕ Sterling, Ill.; Rush Medical College, Chicago, 1926; on the staff of the Public Hospital; aged 39; died, September 22, as the result of an automobile accident.

John Cadwallader Lewis ⊕ Lexington, Ky.; College of Physicians and Surgeons, Medical Department of Columbia College, New York, 1895; aged 66; died, September 4, of heart disease.

George P. Brice, Flowery Branch, Ga.; Atlanta Medical College, 1885; member of the Medical Association of Georgia; aged 75; died, September 1, of pancreatic abscess.

Charles Walden Vandiver, Fort Myers, Fla.; University of Alabama Medical Department, Mobile, 1904; aged 65; died, August 20, in the Lee Memorial Hospital, of embolism.

Clayton M. Mann, East Detroit, Mich.; Western Reserve University Medical Department, Cleveland, 1892; aged 68; died suddenly, September 1, of cerebral hemorrhage.

George Harlan Heilman, Frankfort, Ky.; University of Louisville Medical Department, 1909; aged 56; died, August 24, of cirrhosis of the liver and chronic nephritis.

Frank B. Morgan, Dallas, Texas; Medical College of Indiana, Indianapolis, 1888; aged 68; died, August 27, of hypertrophy of the prostate and arteriosclerosis.

Hugh L. Taylor ⊕ Denver; Denver College of Medicine, 1891; fellow of the American College of Surgeons; aged 65; died in July at the Mercy Hospital.

Thomas Jefferson Rice, Tigrett, Tenn.; University of Nashville Medical Department, 1868; Confederate veteran; aged 94; died, August 29, of uremia.

Lewis Scott Knisely, Fort Sumner, N. M.; University of Kansas School of Medicine, 1934; aged 29; died, September 29, at Clinton, Mo., of tuberculosis.

Joseph D. Sullivan, Milwaukee; Marquette University School of Medicine, Milwaukee, 1914; aged 56; died suddenly, September 4, of heart disease.

Charles Jay Logan, Socorro, N. M.; University of Vermont College of Medicine, Burlington, 1888; aged 68; died, July 22, of coronary thrombosis.

William Edgar Kessinger, Pomona, Calif.; Kentucky School of Medicine, Louisville, 1885; aged 79; died, August 9, of coronary occlusion.

William Robert Welch, Denver; Hahnemann Medical College and Hospital, Chicago, 1889; aged 78; died, July 19, of angina pectoris.

John H. Tompkins, Baltimore; Leonard Medical School, Raleigh, N. C., 1896; aged 67; died, July 19, of chronic nephritis.

CORRECTION

The statement in the obituary of Dr. George Frederick Stericker of Springfield, Ill., in THE JOURNAL, October 19, page 1288, that he was "past president and secretary of the Sangamon County Hospital" was an error. A son, Dr. George B. Stericker of Springfield, was formerly secretary of the Sangamon County Medical Society.

Bureau of Investigation

HAINES' GOLDEN TREATMENT

A Long-Lived Medical Swindle Debarred from the Mails

A particularly heartless swindle that has been operated through the United States mails for over fifty years has finally had the mails closed to it. On August 26, 1935, the Postmaster General, on evidence satisfactory to him, declared that the Dr. J. W. Haines Company, the Golden Specific Company and their officers and agents as such of Cincinnati, Ohio, were engaged in conducting a scheme for obtaining money through the mails by means of false and fraudulent pretenses, and that in view of this, the postmaster at Cincinnati was instructed to stamp "Fraudulent" and return to the writers all letters addressed to the concerns named and refuse to pay any postal money order drawn to the order of the concerns or parties involved.

The "Dr. Haines Golden Treatment" used to be known as the "Golden Specific" before lying on or in the trade package carried with it a risk of prosecution. For over half a century mothers, wives and sisters have been led to believe that they can cure their loved ones of the drink habit by the secret administration of the Haines Golden Treatment. Never in all this period has a case of drunkenness been cured by such means. But swindlers who exploit "liquor cures" know full well that in the very nature of the case the victims who learned that they had been defrauded would never complain because of the publicity that is inseparable from such complaints. Next to the vampires who used to sell narcotic mixtures under the specious claim that they would cure the drug habit, the most heartless swindlers in the "patent medicine" field are the exploiters of preparations sold under the claim that the drugs may be administered without the knowledge of the patient and yet will cure the patient of the drink habit.

Eighteen years ago—October 27, 1917—there was published in this department of THE JOURNAL an article on the Haines Golden Treatment in which it was characterized as "a cruel humbug." In the article there were given the results of an analysis made by the A. M. A. Chemical Laboratory, showing that the alleged cure was essentially a mixture of milk sugar and starch, with a small amount of red pepper and a minute amount of ipecac.

A year previously (1916) the state chemists of North Dakota analyzed the same preparation and reported that the stuff consisted of about 75 per cent of milk sugar, 15 to 20 per cent of wheat starch, with ipecac, capsicum and cascara present. There was no cascara found by the chemists of the American Medical Association, but the composition of nostrums is notoriously variable.

In November, 1929, the National Better Business Bureau issued a special bulletin to periodical publishers on the Haines Golden Treatment, pointing out the fraudulent claims made for the product and stating that the exploitation of the Golden Treatment was contrary to the public welfare. In the same year the Federal Trade Commission ordered a complaint against the Dr. J. W. Haines Company, because the concern was advertising that its compound would cure a person of the liquor habit without the patient's knowledge. The Commission proceeded against the Haines concern, charging that the representations and claims made were false and misleading and deceived and misled the purchasing public. Two years later the Haines concern entered into a stipulation with the Federal Trade Commission to correct and revise its advertising copy, its literature and form-letters so as to eliminate all the objectionable material. The Commission reported that the concern had agreed that it would not at any time in the future publish or distribute such objectionable representations or "any statement, representation or implication that is equivalent or similar in form or in substance to those eliminated" from the advertising material. Of course, a quackish concern of this type could hardly be expected to keep its word, and, in fact, it was still making the fraudulent claim that the Commission had objected to up as late as the time that the Post Office Department proceeded against them.

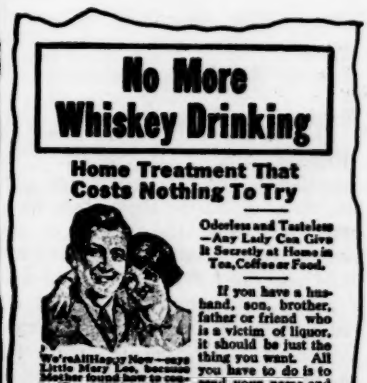
The Haines preparation could not be reached under our present inadequate Food and Drugs Act, because the exploiters were shrewd enough not to make false or fraudulent claims on or in their trade package. They confined their lying to the newspapers and cheap magazines that would accept their advertisements—and the Pure Food and Drugs Act does not penalize fraudulent newspaper advertising.

The Hon. Karl A. Crowley, Solicitor of the Post Office Department, in his memorandum to the Postmaster General recommending the issuance of a fraud order, brings out some interesting facts regarding this long-lived swindle: According to the memorandum, the sole owner of the business at the present time is one Richard J. Goettle, Jr. The fraud was "first launched in 1882 at Cincinnati, Ohio, by J. M. Boyle and Dr. Haines, who later sold out his interest to Mr. Boyle." As far as we have been able to find out, there never was a Dr. J. W. Haines in Cincinnati.

It seems that in 1913 Boyle sold the business to the father of the present owner. Richard J. Goettle, Jr., has operated the fraud since his father's death in 1931.

The Solicitor's memorandum states further that, according to Goettle's own claim, the annual gross receipts have approximated $78,000! Of this, 95 per cent was derived from mail-order sales and about 5 per cent from drug store sales. The mail-order sales were promoted by advertisements that were published in about a hundred daily newspapers and about fifty magazines—publications that were willing to share the profits of this fraud in order that the contact could be made between the victims and the swindler. A typical advertisement read in part as follows:

DRUNKENNESS CURED
It is Now Within the Reach of Every Woman to Save the Drunkard.
By a new discovery which can be given in tea, coffee or food. It does its work so silently and surely that while the devoted wife, sister or daughter looks on, the drunkard is reclaimed even against his will and without his knowledge or co-operation. Send your name and address to Dr. J. W. Haines, 1012 Glenn Bldg., Cincinnati, Ohio, and he will mail enough of the remedy free to show how it is used in tea, coffee or food and that it will cure the dreaded habit quietly and permanently, also full directions how to use it, books and testimonials from hundreds who have been cured and everything needed to aid you in saving these near and dear to you from a life of degradation and ultimate poverty and disgrace.
MISS MARY ROBERTS.
The above is a picture of Miss Mary Roberts, 1033 Golden Gate Ave., San Francisco, Cal., who successfully cured her brother after he had led a drunkard's life for years. She most heartily recommends Golden Specific to every woman and wants to do everything in her power to help others save the drunkard.


No More Whiskey Drinking
Home Treatment That Costs Nothing To Try
Odorless and Tasteless —Any Lady Can Give It Secretly at Home in Tea, Coffee or Food.
We're All Happy Now—says Little Mary Lee, because Mother found how to control Papa's Whiskey Drinking (and we want every woman to know about it).
If you have a husband, son, brother, father or friend who is a victim of liquor, it should be just the thing you want. All you have to do is to send your name and address and we will send absolutely FREE, in plain wrapper, a trial package of Golden Treatment. You will be thankful as long as you live that you did it. Address Dr. J. W. Haines Co., 283 Glenn Building, Cincinnati, Ohio.

For fifty years this fraudulent cure has been advertised. Here is a reproduction (reduced) of two typical advertisements. The one on the left appeared about thirty-six years ago (*Cosmopolitan*, December, 1899); the one on the right was published in 1935.

"HE HATES WHISKEY NOW
"AN ODORLESS AND TASTELESS TREATMENT DID IT

"Any lady can give it secretly at home in tea, coffee or food, and it costs nothing to try: If you have a husband, son, brother, father or friend who is a victim of whiskey, beer, or wine, send your name and address to Dr. J. W. Haines Co., 490 Glenn Bldg., Cincinnati, Ohio, and they will send you absolutely free, in plain wrapper, a trial package of this wonderful treatment. What it has done for others is an example of what it should do for you when used as directed. Write today and be thankful all your life."

Those who answered the advertisements were sent the so-called trial package and a form-letter, together with a pamphlet. The form-letter stated that the powder could be given secretly, but that even if the liquor addict knew that he was getting it, "the result would not be changed, because this treatment does not affect his wish, since the purpose of the

preparation is to relieve the craving for liquor . . ." The same letter emphasized:

"No one in your family need know the victim is being given Golden Treatment. The victim himself should never know it, but allowed to believe that he quit of his own free will. Remember that the passion for drink may be handed down by heredity. Aid not only the living man but those who may live after him."

The pamphlet that accompanied the form-letter purported to show the injurious effects of alcohol on the brain, heart, stomach, liver, nose, eyes and kidneys. Lurid colored pictures such as used to be published in the common-school textbooks of the 90's were scattered through the pamphlet.

A question blank also accompanied the form-letter, pamphlet and trial package, which called for the answers to several questions as to age, weight, sex, occupation and habits of the addict. The blank carried a statement that the questions should be carefully answered "in order that we may fully understand condition of patient." The facts were, however, that no attention whatever was paid to the symptom blank and, in fact, there was no one connected with the Haines outfit who was qualified to express an opinion, for, as the memorandum to the Postmaster General brings out, neither Goettle nor anyone connected with the enterprise had any medical training. The memorandum states that the so-called Golden Specific or Golden Treatment contained the following ingredients in powder form:

Milk sugar	Prickly ash bark
Pleurisy root	Angelica root
Bayberry	Red pepper
Ginger	

The so-called treatment came in three strengths—"Mild," "Regular" and "Extra-Strong." They all contained the same ingredients, but the proportions varied. Milk sugar made up the bulk of all of them, but whereas the "Mild" treatment contained about 11.5 grains of milk sugar in the 15-grain powder, the "Extra-Strong" contained 8 grains; while the Mild contained 1 grain of ginger, the Extra-Strong contained 2 grains, and whereas the Mild contained 1/10 of a grain of red pepper, the Extra-Strong contained 1/30 of a grain.

Richard J. Goettle, Jr., the sole owner of this fraud, did not appear at the hearing. He was represented by an attorney and the hearing lasted two days. Goettle's attorney offered no testimony with respect to the efficacy of the so-called treatment. The government called on two physicians, both of whom had had extensive experience in dealing with chronic alcoholics and dipsomaniacs. Both physicians testified, of course, that, in their opinion, the so-called Golden Treatment would not cure the liquor habit or cause drunkards to stop the immoderate use of alcoholic beverages, nor would it make any drinker grow to abhor the very sight or odor of liquor, as Goettle had claimed. Furthermore, they testified that this preparation of ginger, red pepper and other substances would not soothe an inflamed stomach or relieve ulcerations, nor would it steady the nerves or restore the vital organs to normal conditions. The physicians naturally brought out the definite relationship between the mental condition of the liquor addict and his addiction. Goettle's lawyer, in cross-examining one of the medical experts who were testifying for the government, endeavored to establish that while the dosage of drugs in the Golden Treatment was less than the recognized "allopathic" dosage, they were homeopathic dosages. The physicians' testimony established that the treatment was based upon neither homeopathic nor scientific dosages. Furthermore, as the Solicitor's memorandum brings out, dosages of drugs must be adapted to individual requirements at the time of administration, a fact that Goettle was undoubtedly familiar with if he had investigated the subject to the extent which his attorney's brief indicated. Nevertheless, the Golden Treatment was sold in three arbitrarily-fixed strengths to all, without any regard to individual needs.

A brief that Goettle's attorney filed subsequent to the hearing, and which was given due consideration by the Solicitor of the Post Office Department, stated that many testimonials had been received by Goettle from physicians. It appears that some half-dozen letters of this character were submitted at the hearing, but perusal of the letters showed that some were from practitioners who were not physicians but osteopaths or others having little or no training in the use of drugs, and none of the persons apparently was familiar with the formula or the ingredients of the so-called treatment. Still others were evidently anxious to obtain a profit from resale of the treatment to patients. Two of the alleged physicians whose testimonials were submitted were confessed morphine addicts, as well as drunkards. Mr. Crowley states that most of the letters indicated a "serious lack of education on the part of the writers."

Goettle's excuse for producing no witnesses to testify in his behalf was that it was impossible to present medical expert testimony, due to the unwillingness of physicians to testify, because, as he put it, they "are fearful of blacklisting and criticism by a certain minority in their profession who profess to act as their brother's keeper." This charge has always been made by fakers in the medical field. While it is certainly a fact that decent medical men will not sell their services to medical fakers, it is also a fact, as unfortunate as it is notorious that there are a few renegades in the medical profession who can be counted on to testify for a price for even the most notorious of quacks. In discussing Goettle's charge just quoted, the Solicitor for the Post Office Department states that if Haines' Golden Treatment really possessed the merits claimed for it, Goettle would have had no difficulty in getting medical men to testify. Furthermore, Mr. Crowley states that members of the medical profession frequently testify before the fraud order hearings in cases involving medical questions.

Goettle's case consisted chiefly in what purported to be quotations from various medical textbooks, with argumentative interpretations thereof by Goettle's attorney. As Solicitor Crowley pointed out in his memorandum, aside from the fact that Goettle's lawyer was not qualified to interpret such medical texts on a scientific basis, the arguments that were contained in his brief were in many instances based on incomplete and misleading excerpts from the works of medical writers.

The postal authorities are to be commended for having put out of business one of the most despicable forms of fraud in the medical field.

Correspondence

WISCONSIN LAW ON BLOOD TESTS

To the Editor:—In March 1935 Governor Lehman signed a bill granting authority to the courts of New York State to order blood tests in certain cases, such as those involving alleged paternity and interchange of babies. A similar bill in the state of Wisconsin, sponsored by myself, passed both houses and was signed by Governor La Follette in August 1935 (758A, chapter 351, Laws of 1935).

My object in this communication is to put on record in the columns of THE JOURNAL certain features of the Wisconsin law that may prove helpful to those interested in sponsoring similar legislation in other states.

In the first place, this bill distinctly states that in cases of disputed paternity the court may order that "the complainant, her child, and the defendant submit to one or more blood tests to determine whether or not the defendant can be excluded as being the father of the child." This makes it clear that the present value of these tests is only to exclude the possibility of paternity rather than to provide positive proof thereof. In order to prevent further the court, jury and district attorney from wrongly interpreting these tests as proof of paternity, the Wisconsin bill states that "the result of the test shall be receivable in evidence but only in cases where definite exclusion is established."

The Wisconsin law allows tests to be made not only by "duly qualified physicians" but also "by other duly qualified persons." Obviously the bacteriologist or serologist most qualified to perform these tests need not necessarily be trained as a physician.

The law provides that "whenever the court orders such blood tests to be taken and one of the parties shall refuse to submit

to such test, such fact shall be disclosed upon the trial unless good cause is shown to the contrary." The significance of this part of the law is quite evident.

The present Wisconsin law makes provision for the use of the blood tests not only in cases in which the state or the community is a party to the action but also in civil actions in which paternity or maternity is in question, for instance, cases in which the problem is to exclude a false claimant to an estate or to detect alleged interchange of babies.

However, no provision as yet is made to grant authority to the courts for the use of these tests in criminal cases. This aspect was advisedly omitted, since it was felt that successful legislation was more assured with a bill not too involved. Of course, this can be readily corrected by further legislation. The suggestion is here made that workers in other states in framing the proposed bill provide in it also for criminal cases, since in the future a more comprehensive bill is not apt to suffer in its chance for passage, in view of the recent successful legislation in New York and Wisconsin.

PHILIP LEVINE, M.D.,
Beth Israel Hospital,
Newark, N. J.

BISMUTH IN OIL

To the Editor:—In the September issue of the *Journal of the American Pharmaceutical Association* I published an article describing a procedure for evaluating "Bismuth in Oil" preparations and reported in a general way the results obtained on several products. No figures were given nor were the names of any firms mentioned. Recently there has come to my attention an advertisement of the Loeser Laboratory, 22 West Twenty-Sixth Street, New York, in which this article has been reproduced in its entirety, and the implication is made that this is evidence of the superiority of their product. "We offer the following reprint as corroborating evidence of our claim"; then follows the article; then "It is obvious that in Loeser's Bismuth in Oil a new and higher standard has been established for bismuth suspensions" is part of the language employed.

This article was reproduced without my consent or knowledge and without the consent or knowledge of the editor of the *Journal of the American Pharmaceutical Association.* A vigorous protest on my part brought a profuse apology and a statement that their advertising would be confined to isolated paragraphs. A specimen of this mailed to me still retains all the objectionable features. In fairness to all concerned, and particularly to physicians who may have received an incorrect impression, I wish to make it clear that neither the Maryland State Health Department nor I endorse, by implication or in any other manner, the superiority claims of this firm. On the other hand, we deplore the rather questionable and certainly unethical means employed to further this product.

WILLIAM F. REINDOLLAR, Baltimore.
Assistant Chief, Bureau of Chemistry, State of Maryland Department of Health.

GONOCOCCIC TENOSYNOVITIS

To the Editor:—In THE JOURNAL, September 28, is an article on "Acute Suppurative Gonococcic Tenosynovitis" by Walter Birnbaum and C. Latimer Callander. In the bibliography no reference is made to the case described by Isadore Zadek in THE JOURNAL of June 15, on "Gonorrheal Tenosynovitis of the Long Head of the Biceps Brachii." Although in the latter case the diagnosis was not made on the pus, the tissue examined showed numerous pus cells with intracellular gram-negative biscuit shaped diplococci.

ABRAHAM S. ROTHBERG, M.D., New York.

Queries and Minor Notes

ANONYMOUS COMMUNICATIONS and queries on postal cards will not be noticed. Every letter must contain the writer's name and address, but these will be omitted on request.

USE OF POISON GAS BY THE U. S. ARMY

To the Editor:—In an article entitled "Air-Raid Precautions," on page 358 of the August 24 issue of the *British Medical Journal,* I note the following statement: "It is notorious, too, that the U. S. A. favor poison gas as an instrument of warfare, considering it not more inhumane than high explosive, and constantly using it in cases of civil riot." To the best of my knowledge it is tear gas which is favored for such use as being temporarily incapacitating but nontoxic. It occurs to me that the statement of the American Medical Association to the editors of he *British Medical Journal* might bring about a correction of a misstatement which seems unfortunate. ANNA M. WALLACE, M.D., Elizabeth, N. J.

ANSWER.—The assertions quoted from the *British Medical Journal* are incorrect in that they represent that the United States favors poison gas as an instrument of warfare and that the United States uses poison gas in cases of civil riot.

Actually the United States has been obliged to reconcile itself to the likely use of gas in future warfare, following the failure of efforts (in which the United States took prominent part) to proscribe such use by universal agreement.

The use of poison gas in domestic disturbances has never been resorted to within the United States. Chemicals used in riot duty are carefully selected from the group of nonlethal agents. Regrettable misuse of the term "poison gas" results frequently in its application to chloracetophenone and diphenylaminechlorarsine, neither of which is a casualty producer. An attempt to clarify this point was made by the British delegation before the Preparatory Disarmament Commission in 1930, supported by the American representative, Mr. Hugh Gibson, who noted the inconsistency of banning from warfare types of chemicals that were freely and advantageously used in suppressing civil disturbances. However, this attempt by British and American representatives to find a satisfactory solution to a complicated political-technical problem can in no sense be interpreted as committing the United States in favor of the general use of poison gas.

The British Draft Convention, submitted to the General Disarmament Conference, and to which the support of the United States was pledged by President Roosevelt, May 16, 1933, provided for the abolishment of chemical warfare in toto.

PARKINSONISM AND DIABETES

To the Editor:—In the early part of 1929 I detected that I had sugar in the urine, 10 per cent; I immediately put myself under a very strict diet and high dosage of insulin, using sometimes 60 units once a day. Within about a month the sugar and acetone disappeared; however, I continued the treatment for about seven months. Since then I have kept the sugar under control on a moderate diet and the periodic use of insulin. Insidiously with the sugar, a nervous condition in my left foot and arm gradually and imperceptibly crept along, such as paresthesia in the left foot, edema in the left ankle, and dull pain in the left arm, together with a labored movement, heaviness, and slow execution in the movements in these limbs. On exertion and effort there is a tremor; there is also muscular pain and stiffness. I attributed this to the sugar, neuritis, and muscular rheumatism. Recently I have consulted some specialists on nervous diseases. They made the diagnosis of parkinsonism (not Parkinson's disease). What is the etiology of parkinsonism? Is there any relation in the causative factor of sugar and parkinsonism or is the relation merely incidental in my case? Has a severe strict diet or the continued high dosage of insulin anything to do with parkinsonism? What is the difference between Parkinson's disease and parkinsonism? What is the best line of treatment in parkinsonism? Is hormone therapy of any use? M.D., Egypt.

ANSWER.—Parkinson's disease (paralysis agitans) may be defined as a chronic progressive disease of the central nervous system, characterized by muscular rigidity, weakness, slowness of movement, and rhythmic, spontaneous tremor. This seldom occurs before the fiftieth year of age and, while no definite pathologic changes have been established, it is believed to be due to degenerative vascular changes affecting the lenticular nucleus.

Parkinsonism refers to a similar syndrome, which may occur at younger age periods and which is due to other causes. The description is consistent with this diagnosis. There are no treatises confined to this subject, but there is a large body of literature on epidemic encephalitis and carbon monoxide poisoning, both of which may be followed by parkinsonism.

There is no established relationship between diabetes and parkinsonism, although, in view of the newer work on the relation

of the hypophysis to diabetes, it might be worth while seeking for a common cause that might affect the nervous ganglions and this gland. It does not seem likely that either the strict diet or insulin dosage could have anything to do with parkinsonism. No hormone therapy for parkinsonism is known. The usual treatment is entirely symptomatic. The tinctures of hyoscyamus, stramonium or scopolamine are frequently employed, as well as calcium and parathyroid.

STREPTOCOCCIC PNEUMONIA

To the Editor:—A physician, aged 42, was taken ill, April 19, while making a call. He had a chill followed by a temperature of 103 F. and pain of the lower lobe of the left lung, with coughing and expectoration of blood-stained mucus. He continued to grow worse. There were daily chills, severe sweats and an increase in temperature, with signs of cyanosis. Pain of the lower lobe of the left lung continued, with pain more severe over the heart. I was called to see him on the afternoon of April 21. Examination of the chest showed consolidation of the left lower lobe with a spreading of infection into the bronchial tubes and the left and right lungs. The pulse was 108, the temperature was 101, and respirations numbered 24. At this time he had not had his daily chill with increase in temperature. Blood and sputum cultures were positive for Streptococcus viridans and a hemolytic staphylococcus infection. No pneumococcus was found; examinations were made by the state laboratory. Each afternoon between 2 and 4 the patient would have a severe chill, increase in temperature, and an attack with pain over the heart and cyanosis. The cough and expectoration of blood and mucus continued to grow worse. On the night of April 22, all treatment was discontinued and he was placed in an oxygen tent sent out by the Ohio Chemical Company. A mixture of oxygen 93 per cent and carbon dioxide 7 per cent was given for ten hours. From this time he was given a mixture of straight oxygen, 12 liters a minute, and morphine for pain, and showed some improvement. In addition to this I advised 1,000 cc. of 10 per cent dextrose and physiologic solution of sodium chloride intravenously daily and a blood transfusion if the infection continued to spread. White blood count April 23 showed 17,500, polymorphonuclears 90 per cent, temperature 97.6. He died on the morning of the 26th, following acute dilatation of the stomach. He received only 1,500 cc. of dextrose and saline solution. He did not receive a blood transfusion. Please advise me, as we are having a sporadic epidemic of this type of pneumonia here. Would you have advised a blood transfusion? If so, how soon after the diagnosis was made? Would you have advised daily 1,000 cc. of dextrose and saline solution intravenously? If so, how soon? Would you have advised some form of streptococcus serum or a nonspecific serum? What other treatment would you have advised in addition to the foregoing, if any? Please omit name.

M.D., Alabama.

ANSWER.—Obviously the patient had an overwhelming streptococcic pneumonia with bacteremia. It is unlikely that it would have terminated favorably as a result of any known type of management. There is no known serum or vaccine available the use of which would have materially influenced the outcome. The prompt administration of oxygen is to be commended. To place the patient in an atmosphere supersaturated with oxygen (from 60 to 70 per cent), preferably by means of an oxygen chamber, but lacking that, an oxygen tent carefully adjusted, or the nasal catheter method, is the most effective clinical procedure available. Morphine is indispensable. Intravenous dextrose solution, from 5 to 10 per cent, given slowly, may well be administered daily in amounts of from 1,000 to 2,000 cc. unless contraindicated. Continuous gastric siphonage and blood letting are frequently of great benefit in acute pneumonia. Blood transfusions would probably be of little avail in an acute overwhelming pneumonia, such as is related in this case, and is probably not to be recommended. The use of nonspecific serums and vaccines in acute pneumonia is a question not settled as yet. There are those who enthusiastically recommend and use them, citing prompt temporary and occasionally permanent remissions in clinical manifestations. In a pinch, lacking other means better supported by clinical observation, such procedures may be and often are used. There can be no criticism of so doing when employed under such circumstances.

TREATMENT OF STERILITY

To the Editor:—Two young married women have been married several years and have never been pregnant. Both are anxious to have children. One of them has a decided acid vaginal secretion, which I am trying to neutralize partly. There is no history of tubal inflammation and they are as passionate as the normal woman. Is there anything in the realm of glandular therapy that would help them? I get a bewildering number of pamphlets extolling the different products. If there is anything worthy of trial will you kindly let me know? Please omit name.

M.D., West Virginia.

ANSWER.—The answer to these questions is not to be found in the advertisements of pharmaceutical concerns but may be revealed after a thorough investigation of the matings in question. A scientific inquiry into the fecundity of each partner and the performance of accepted tests for fertility will doubtless uncover the cause or causes of the reproductive failure.

A plan for making such a study of apparently infertile couples will be found in recent books on sterility and in the current literature.

Acidity of the vaginal secretion is the normal reaction and as such requires no treatment.

Glandular therapy may be beneficial to one or the other partner, but a valid indication for its use must first be established. Following are references:

Meaker, S. R.: Human Sterility, Baltimore, Williams & Wilkins Company, 1934.
Stein, I. F., and Leventhal, M. L.: Infertility and Sterility, THE JOURNAL, Feb. 20, 1932, p. 621.
Bland, P. B.; First, Arthur, and Goldstein, Leopold: The Clinical Investigation of Functional Sterility in the Female, THE JOURNAL, Oct. 19, 1935, p. 1231.
Titus, Paul: Sterility, THE JOURNAL, Oct. 19, 1935, p. 1237.

GRATING NOISE IN CERVICAL VERTEBRA

To the Editor:—An unmarried white woman, aged 42, complains of a grating noise, which she believes comes from the region of the upper cervical vertebra and base of the brain. This is noticed particularly when she rotates her head from side to side but does not bother her when she moves her head in the up and down direction. The noise can be heard without the aid of a stethoscope when the examiner places his ear near the occipital and mastoid regions of the head. This noise is much like that which is heard on bending a knee that has long been afflicted with a chronic hypertrophic type of arthritis. The patient dates the beginning of this disturbance back to four years ago following a course of sweat baths for a hypertension. Except for this hypertension, which varies from 160 to 190 systolic with a diastolic pressure of 90 mm. of mercury, her physical condition is excellent. There is no evidence of any condition of arthritis in any other part of the body and, so far as I can ascertain, there are no foci of infection. Anteroposterior and lateral roentgenograms of the cervical spine are negative. This disturbance has become quite an annoyance to the patient, and any suggestions you may have to offer in the way of therapy will be greatly appreciated.

HARVEY L. JORGENSON, M.D., Marinette, Wis.

ANSWER.—This patient should be treated as though she had arthritis. There are practically always two factors involved in this lesion: viz., trauma (either a single severe one or multiple minimal ones), and infection especially of the teeth, throat, sinuses, gastro-intestinal and genito-urinary tracts and lymphatic system.

Has her neck ever been treated by an osteopath or chiropractor?

If the condition is sufficiently annoying, the following treatment is outlined:

1. Rest on a hard mattress, in a bed with its head elevated from 6 to 8 inches, with
2. Application of head traction.
3. Immobilization in a plaster-of-paris cast or a Schrock type of brace, followed by a special collar like the collars of H. O. Thomas, Schanz or Wallace.
4. Physical therapy consisting of radiant heat, massage and either diathermy or short wave diathermy.

Foci of infection and metabolic or endocrine disturbances should be investigated. Vaccine may be considered.

TREATMENT OF AMENORRHEA

To the Editor:—A white girl, nearly 18 years old, has not yet menstruated. She complains of occasional pains in the back, headaches and fatigue. The past history is negative. She weighs 125 pounds (56.7 Kg.), is 5 feet 5 inches (165 cm.) tall and is of normal development. The thyroid is not palpable. The blood count is normal. The basal metabolism is normal. What is the cause and what treatment should be given? I have given her four injections of antuitrin-S, 100 rat units each time. Is it advisable to use theelin simultaneously and for how long a period? Should the dose be gradually increased and in what intervals? Rectal examination does not reveal anything abnormal. Vaginal examination is impossible. Please omit name and address in case of publication.

M.D., New York.

ANSWER.—Primary amenorrhea is usually difficult to overcome. In some instances menstruation sets in spontaneously, especially in those under 20 years of age. In many cases of amenorrhea, bleeding may be induced by giving huge amounts of estrogenic substance; this may be followed (but not necessarily) by corpus luteum hormone (progestin). However, these bleedings will recur only as long as the former or both hormones are administered. If the hormones are not given, the so-called menstrual periods cease. Since ovulation is not produced, there is generally no use in giving this treatment even to married women, because pregnancy cannot possibly be brought about by it. In the case cited undoubtedly the patient has a hypoplastic uterus. While such a uterus can be enlarged by means of hormone therapy and develop a functioning endometrium, it will retrogress as soon as the hormone therapy is stopped. Not infrequently desiccated thyroid is much more helpful in such cases than other hormone therapy.

The only value there is to the bleeding induced by hormones is a psychic one. If women who have amenorrhea are convinced that they are as healthy as women who do menstruate, there is usually no need to treat them for the amenorrhea.

If the physician would like to try hormone therapy he may administer 50,000 international units of estrogenic substance (Amniotin, theelin and so on) on the first, fourth, seventh, tenth and thirteenth day of a course and then give 15 rabbit units of progestin on the seventeenth, eighteenth, nineteenth, twentieth and twenty-first days. If bleeding is to follow, it will usually take place within ten days after the last injection of progestin.

Anterior pituitary-like gonadotropic hormone of the urine of pregnant women may be administered in place of progestin, but it is doubtful whether the result obtained is equivalent. This hormone should be injected intramuscularly in doses of from 150 to 200 rat units daily for five or six injections. In order to produce successive periods of bleeding, these courses of treatment will have to be repeated.

ARTHRITIC ANKLE

To the Editor:—A man, aged 74, married, came to me with the complaint of aching ankle joints of about three months' duration, aggravated by walking but present during rest as well. The ache, or a "weakness in the ankles," is localized in the ankle joints. Examination of the feet did not disclose anything significant; the dorsalis pedis and posterior tibial arteries were palpable; there was no cyanosis or trophic changes of any kind; there was no evidence of arthritic changes, the motion of the joint was unimpeded, and no crepitus was elicited. The longitudinal and transverse arches were apparently normal. He used arch supports owing to the fact that his occupation necessitates his standing nearly all day. The general physical examination revealed a blood pressure of 180 systolic, 88 diastolic; temperature, 98.2 F.; pulse, 50; respirations, 18. His hearing was impaired. The tympanic membrane showed retraction, and there was distortion of a cone of light. The pupils were equal and regular and reacted to light and in accommodation. Examination of the fundus revealed tortuosity and irregularity of the caliber of the vessels. The teeth were all removed and supplanted by plates. The tonsils were atrophic. The sinuses were apparently normal by transillumination. The lungs were essentially normal. The heart was enlarged downward and outward, and auscultation revealed accentuation of the second pulmonic sound. No murmurs were noted: the rhythm was normal except for occasional extrasystoles and the slow rate already noted. The abdomen was essentially normal, as were the reflexes. Undoubtedly the patient's occupation has much, if not everything, to do with his complaint. For economic reasons he cannot give up his work. He has consulted several physicians in the last few months, without relief. Besides the arch support that he is using at present he has used for longer or shorter intervals ankle supports, diathermy and strapping. Roentgenograms, which I have not seen, have not revealed any pathologic changes in the involved joints. Your comments and suggestions for further treatment would be greatly appreciated.

S. J. WOJCIK, M.D., Paw Paw, Ill.

ANSWER.—While the lesion seems to be in the ankle joint proper, it may be in the subastragalar joint. One may have an arthritic joint without positive pathologic changes in the roentgenogram.

The patient's weight is not stated, nor is mention made of the basal metabolic rate or his dietary habits.

The various factors that have to be eliminated in this case are mechanical, circulatory, traumatic, infectious, myogenic, neurogenic, metabolic and endocrine. The chief traumatic factors may be occupational.

A few days of absolute rest in bed with the application of an anodyne lotion and fomentations may be helpful. Proper high laced shoes and resilient supports with a slightly raised inner heel border may be all that is required to give the man comfort. In addition, massage and contrast sprays should improve the circulation. If unsuccessful, a plaster cast and crutches for a short period would be indicated.

MULTIPLE SCLEROSIS

To the Editor:—What is the latest treatment for multiple sclerosis? Sodium cacodylate, quinine and injection of whole blood intramuscularly were used, with no evidence of benefit.

M.D., Michigan.

ANSWER.—The latest treatment for multiple sclerosis remains as yet empirical. Any disease that has spontaneous remissions usually boasts of multiple and variegated types of treatment. In a number of cases, remissions do not occur at all. The following is suggested: Absolute bed rest for a period of from six to ten weeks for an acute case; this rest includes no reading. During the chronic period, abstention from all forms of severe exercise together with solution of potassium arsenite in doses of from 0.2 to 0.6 cc. three times a day, neoarsphenamine intravenously once a week or quinine hydrochloride, starting with 0.03 Gm. three times a day, gradually increased until about 0.3 Gm. three times a day is taken. For acute retrobulbar neuritis, in addition to absolute rest one should give foreign protein intravenously (typhoid paratyphoid) for at least six or seven injections. The most important part of any regimen in the management of a case of multiple sclerosis is absolute rest and more rest.

COMPLICATIONS IN PNEUMONIA

To the Editor:—Please inform me as to the proper treatment of the following complications in an otherwise typical case of lobar pneumonia: A toxic hepatitis (manifested by persistent and gradually increasing jaundice of skin and sclerae, stupor and other nervous manifestations accompanying jaundice. The liver apparently not enlarged or tender to palpation). Persistent, exhausting and uncontrollable hiccups. The patient died, but the heart and circulation remained good almost to the end. Several years ago, I am informed by the patient's relatives, the patient had a rather severe case of malaria. Do you think that the malaria could have so injured the liver that it failed to function normally when obliged to take care of the toxin due to the pneumonia? In other words, how much of a part would malaria play as an etiologic factor in the causation of a toxic hepatitis accompanying lobar pneumonia? Dextrose intravenously was given every day, once a day 50 cc. of concentrated solution and, toward the last, three or four times a day. Please omit name and address.

M.D., North Carolina.

ANSWER.—Jaundice is not a rare complication in pneumonia, especially in severe infections. In the case described the toxic hepatitis and stupor strongly suggest pneumococcic bacteremia, a frequent finding in fatal cases of pneumonia. The hiccups may have been another expression of severe toxemia or possibly the result of diaphragmatic irritation (diaphragmatic pleurisy). It is doubtful whether the attack of malaria several years before the pneumonia played any part in the development of the jaundice.

TREATMENT OF LATENT SYPHILIS

To the Editor:—What plan of treatment would you advise for a 53 year old patient, with a 4 plus Wassermann reaction (during a routine test) with no symptoms or physical anomalies? He has had no treatment whatever and the history of infection dates back eighteen years. Kindly omit name and address.

M.D., Chicago.

ANSWER.—In the absence of definite signs of cardiovascular disease or neurosyphilis, the case may be treated as one of latent or asymptomatic syphilis. The patient should receive alternating courses of one of the arsphenamines intravenously and an insoluble bismuth compound intramuscularly with iodides between courses. Treatment should be continued with short periods of rest for at least a year and then intermittently until the positive reaction is reversed.

VENT DISEASE OF RABBITS

To the Editor:—Can you tell me what vent disease is in a rabbit? It is communicable from male to female and vice versa and is characterized by excoriated genitalia and a discharge green in character. Is the microorganism known? What can be done to treat it?

EDWARD N. TURKUS, M.D., Long Beach, Calif.

ANSWER.—Vent disease, or treponemiasis, is caused by the spirochete Treponema cuniculi. This organism is regularly transmitted through the process of breeding from male to female and vice versa. In commercial rabbitries where valuable breeding animals are maintained detailed systematic treatment may be justified. Local applications on the genitalia of a 2 per cent solution of copper sulphate, zinc ointment, or mercurial ointment is recommended. The use of neoarsphenamine for subcutaneous injection in doses varying from 0.05 to 0.15 Gm., according to the size of the animal, may result in a permanent cure if used in conjunction with local applications. Apparently the same line of treatment as is used for the treatment of syphilis in human beings is effective, but recovery may be expected in a shorter time. In ordinary rabbits used for laboratory experimental work such procedures are not justifiable, and it is more economical to dispose of the affected stock, and after a thorough cleansing and disinfection of the hutches to replace them with healthy animals.

FORMULA FOR ECZEMA OF AUDITORY CANAL

To the Editor:—On page 496 (February 9) of THE JOURNAL is the following formula for the treatment of eczema of the auditory canal:

Oil of birch tar	0.8 cc.
Resorcinol	0.4 cc.
Liquid petrolatum	sufficient to make 16 cc.

Our druggist states that he has tried to mix this but that the tar and the resorcinol will not go into solution. Can you advise?

M.D., Minnesota.

ANSWER.—An expert pharmacologist has wrestled with this problem and says that the only method he can suggest is to dissolve resorcinol 0.4 Gm. in glycerin 5 cc., dissolve the oil of birch tar 0.8 cc. in liquid petrolatum 11 cc., then mix the two solutions, and label them "Shake well immediately before using."

BURNING PAIN DURING INTERCOURSE

To the Editor:—A man, aged 55, in perfect health as far as I can find out, lives on a farm. He is 5 feet 11 inches (180 cm.) tall and weighs 200 pounds (90 Kg.). The heart and lungs are normal. The teeth are very bad. The blood pressure is 115 systolic, 60 diastolic. The Wassermann reaction is negative. The blood count is normal. The kidneys are normal. Urinalysis gives negative results. He has some lancinating pains in the lower limbs and vitiligo of the dorsal surface of both hands. He states that during sexual intercourse the semen burns like a red hot poker and if a drop of it touches his wife anywhere on the vaginal mucous membrane it burns her just like fire. I would be glad to know the cause of this if possible. Please omit name.

M.D., Illinois.

ANSWER.—Burning pain during ejaculation is not a rare condition and is generally due either to an intense congestion of the prostatic urethra or to ulcerations or granulations or similar pathologic conditions in that region. Treatment consists in getting rid of the pathologic condition, which is usually accomplished by gentle prostatic massage and instillations of weak (from 1 : 3,000 to 1 : 500) silver nitrate solutions into the prostatic urethra with the Bangs sound syringe.

There is no condition of the ejaculatory fluid that could possibly cause any burning when brought in contact with the vaginal mucous membranes. There may of course be present an intense vaginitis or increased sensibility of the vaginal mucous membrane, but, what is more likely, the symptoms in the wife are either psychic or suggestive.

DEAFNESS AFTER TETANUS ANTITOXINS

To the Editor:—A patient became deaf after receiving tetanus antitoxin. Can tetanus or tetanus antitoxin cause deafness? Kindly do not use my name.

M.D., Michigan.

ANSWER.—A moderately extensive but not exhaustive search of sources of information does not disclose any report in which deafness has been caused by tetanus. Optic neuritis and peripheral neuritis have been noted as occasional complications, and it would accordingly appear not impossible for deafness also to occur after tetanus.

Other possibilities must be considered. Tetanus may occur in a patient who already has auditory changes that have escaped notice of the patient previous to his acute illness. Coexistent disease of the central nervous system, such as cerebrospinal syphilis, also must be considered.

Serum disease frequently follows the injection of foreign serum, especially when large doses, usually required in tetanus, are used. Urticaria as well as hemorrhages in various parts of the body, including joints and internal organs, are frequent, and if such a lesion occurred in the ear, deafness either temporary or permanent might result.

EFFECTS OF SCARLET FEVER TOXIN WHEN SWALLOWED

To the Editor:—A physician's son, aged 6 years, was discovered by his mother playing with a box containing 100 vials of Squibb's scarlet fever toxin. She suspects that the vials were full of solution originally but has no way of being sure. If the child did swallow the contents of all or part of these vials, what may develop, and what would you suggest? Please omit name.

M.D., Vermont.

ANSWER.—If the child was previously susceptible to scarlet fever and swallowed such an amount of toxin he has partially or completely immunized himself against the disease. It will do no harm. No treatment is indicated.

EFFECTS OF AMYTAL

To the Editor:—Please tell me whether the prolonged use of sodium amytal, from 12 to 15 grains (0.8 to 1 Gm.) daily, (1) has a cumulative toxic effect, (2) affects the eyes, (3) results in a habit formation that is difficult to break or (4) is apt to cause skin manifestations. Please omit name.

M.D., New York.

ANSWER.—1. It may have.
2. There is apt to be dilatation of the pupil.
3. Yes.
4. It is.

LIGHT RAYS IN TUBERCULOSIS

To the Editor:—Are actinic rays, artificially produced (such as those generated by a quartz burner of the Hanovia Chemical Company) contraindicated in pulmonary tuberculosis? Would a gradual tanning of the skin of the chest be detrimental or beneficial?

J. PHILLIPS EDMUNDSON, M.D., Kansas City, Mo.

ANSWER.—No, it is not contraindicated but found to be of no value and may do harm in overdosage.

Gradual tanning of the skin of the chest would have no action at all, either detrimental or beneficial.

Medical Examinations and Licensure

COMING EXAMINATIONS

AMERICAN BOARD OF DERMATOLOGY AND SYPHILOGY: *Written examination for Gorup B applicants* will be held in various cities throughout the country, March 14. *Oral examination for Group A and B applicants* will be held in Kansas City, Mo., May 11-12. *Applications for written examination should be filed with the secretary before Jan. 15.* Sec., Dr. C. Guy Lane, 416 Marlboro St., Boston.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY: Written examination and review of case histories of Group B applicants will be held in various cities of the United States and Canada, Dec. 7. *Applications must be filed not later than Nov. 1.* Sec., Dr. Paul Titus, 1015 Highland Bldg., Pittsburgh (6).

AMERICAN BOARD OF OPHTHALMOLOGY: St. Louis, Nov. 18. Asst. Sec., Dr. Thomas D. Allen, 122 S. Michigan Ave., Chicago.

AMERICAN BOARD OF ORTHOPAEDIC SURGERY: St. Louis, Jan. Sec., Dr. Fremont A. Chandler, 180 N. Michigan Ave., Chicago.

AMERICAN BOARD OF OTOLARYNGOLOGY: Kansas City, Mo., May 9. Sec., Dr. W. P. Wherry, 1500 Medical Arts Bldg., Omaha.

AMERICAN BOARD OF PEDIATRICS: St. Louis, Nov. 20. Sec., Dr. C. A. Aldrich, 723 Elm St., Winnetka, Ill.

AMERICAN BOARD OF PSYCHIATRY AND NEUROLOGY: New York, Dec. 30. Sec., Dr. Walter Freeman, 1726 Eye St., N. W., Washington, D. C.

AMERICAN BOARD OF RADIOLOGY: Detroit, Dec. 1-2. Sec., Dr. Byrl R. Kirklin, Mayo Clinic, Rochester, Minn.

ARKANSAS: *Basic Science.* Little Rock, Nov. 4. Sec., Mr. Louis E. Gebauer, 701 Main St., Little Rock. *Medical (Regular).* Little Rock, Nov. 12. Sec., State Medical Board of the Arkansas Medical Society, Dr. A. S. Buchanan, Prescott. *Medical (Eclectic).* Little Rock, Nov. 12. Sec., Dr. Clarence H. Young, 207½ Main Street, Little Rock.

CALIFORNIA: *Reciprocity.* Los Angeles, Dec. 4. Sec., Dr. Charles B. Pinkham, 420 State Office Bldg., Sacramento.

CONNECTICUT: *Medical (Regular).* Hartford, Nov. 12-13. *Endorsement.* Hartford, Nov. 26. Sec., Dr. Thomas P. Murdock, 147 W. Main St., Meriden. *Medical (Homeopathic).* Derby, Nov. 12. Sec., Dr. Joseph H. Evans, 1488 Chapel Street, New Haven.

FLORIDA: Tampa, Nov. 11-12. Sec., Dr. William M. Rowlett, Box 786, Tampa.

KANSAS: Topeka, Dec. 10-11. Sec., Board of Medical Registration and Examination, Dr. C. H. Ewing, 609 Broadway, Larned.

KENTUCKY: Louisville, Dec. 3. Sec., Department of Health, Dr. A. T. McCormack, 532 W. Main St., Louisville.

MAINE: Portland, Nov. 12-13. Sec., Board of Registration of Medicine, Dr. Adam P. Leighton Jr., 192 State St., Portland.

MARYLAND: *Medical (Regular).* Baltimore, Dec. 10-13. Sec., Dr. John T. O'Mara, 1211 Cathedral St., Baltimore. *Medical (Homeopathic).* Baltimore, Dec. 10-11. Sec., Dr. John A. Evans, 612 W. 40th St., Baltimore.

MASSACHUSETTS: Boston, Nov. 12-14. Sec., Board of Registration in Medicine, Dr. Stephen Rushmore, 413 State House, Boston.

NATIONAL BOARD OF MEDICAL EXAMINERS: *Part III.* Boston, Nov. 5-7. Ex. Sec., Mr. Everett S. Elwood, 225 S. 15th St., Philadelphia.

NEBRASKA: Lincoln, Nov. 19-20. Dir., Bureau of Examining Boards, Mrs. Clark Perkins, State House, Lincoln.

NEVADA: Carson City, Nov. 4. Sec., Dr. Edward E. Hamer, Carson City.

OHIO: Columbus, Dec. 3-5. Sec., State Medical Board, Dr. H. M. Platter, 21 W. Broad St., Columbus.

OKLAHOMA: Oklahoma City, Dec. 11. Sec., Dr. James D. Osborn Jr., Frederick.

OREGON: *Basic Science.* Portland, Nov. 16. Sec., Mr. Charles D. Byrne, University of Oregon, Eugene.

SOUTH CAROLINA: Columbia, Nov. 12. Sec., Dr. A. Earle Boozer, 505 Saluda Ave., Columbia.

TEXAS: Houston, Nov. 18-20. Sec., Dr. T. J. Crowe, 918 Mercantile Building, Dallas.

VIRGINIA: Richmond, Dec. 11-13. Sec., Dr. J. W. Preston, 28½ Franklin Rd., Roanoke.

WEST VIRGINIA: Huntington, Oct. 28. State Health Commissioner, Dr. Arthur E. McClue, Charleston.

West Virginia July Report

Dr. Arthur E. McClue, state health commissioner, reports the oral and written examination held at Clarksburg, July 8-10, 1935. The examination covered 11 subjects and included 110 questions. An average of 80 per cent was required to pass. Twenty-five candidates were examined, all of whom passed. Fourteen physicians were licensed by reciprocity and 1 physician was licensed by endorsement. The following schools were represented:

PASSED School	Year Grad.	Per Cent
College of Medical Evangelists	(1934)	86
Howard University College of Medicine	(1934)	85.5
Emory University School of Medicine	(1934)	86.5, 87.7
Rush Medical College	(1935)	86, 87.5
Indiana University School of Medicine	(1934)	85.2
University of Louisville School of Medicine	(1934)	84.9, 86.3, 86.5, 87.2
Johns Hopkins University School of Medicine	(1934)	89.1
University of Maryland School of Medicine and College of Physicians and Surgeons	(1934)	84.7
Tufts College Medical School	(1929)	88.4
New York University, University and Bellevue Hospital Medical College	(1933) 86.1, (1934)	86.7
University of Tennessee College of Medicine	(1931)	81.4
Medical College of Virginia	(1934)	85.8, 87.5, 87.5, 89.4, 89.6, (1935) 86.7
University of Virginia Department of Medicine	(1934)	87.5
Dalhousie University Faculty of Medicine	(1935)	86.7

School	LICENSED BY RECIPROCITY	Year Grad.	Reciprocity with
Northwestern University Medical School		(1929)	Illinois
University of Illinois College of Medicine		(1933)	Illinois
University of Indianapolis Medical Department		(1902)	Indiana
State University of Iowa College of Medicine		(1931)	Iowa
University of Maryland School of Medicine and College of Physicians and Surgeons		(1933)	Maryland
Med. College of Virginia (1930), (1932), (1933), (1934, 2)			Virginia
University of Virginia Department of Medicine (1927), (1929), (1932), (1934)			Virginia

School	LICENSED BY ENDORSEMENT	Year Grad.	Endorsement of
University of Louisville School of Medicine		(1933)	N. B. M. Ex.

Arizona April Report

Dr. J. H. Patterson, secretary, Arizona State Board of Medical Examiners, reports the oral and written examination held in Phoenix, April 2-3, 1935. The examination covered 10 subjects and included 100 questions. An average of 75 per cent was required to pass. One candidate was examined and passed. Four physicians were licensed by reciprocity and 1 physician was licensed by endorsement. The following schools were represented:

School	PASSED	Year Grad.	Per Cent
School of Medicine of the Division of the Biological Sciences		(1935)	80.7

School	LICENSED BY RECIPROCITY	Year Grad.	Reciprocity with
University of Arkansas School of Medicine		(1927)	Arkansas
College of Medical Evangelists		(1930)	California
Stanford University School of Medicine		(1929)	California
State University of Iowa College of Medicine		(1931)	Iowa

School	LICENSED BY ENDORSEMENT	Year Grad.	Endorsement of
St. Louis University School of Medicine		(1933)	N. B. M. Ex.

Book Notices

Diseases of the Thyroid Gland. By Arthur E. Hertzler, M.D., Professor of Surgery, University of Kansas. With a chapter on Hospital Management of Goiter Patients by Victor E. Chesky, M.D., Chief Resident Surgeon, Halstead Hospital. Third edition. Cloth. Price, $7.50. Pp. 348, with 181 illustrations. St. Louis: C. V. Mosby Company, 1935.

This book includes chapters under the classifications of etiology, normal morphology, goiter in childhood, nontoxic colloid goiter, toxic colloid goiter, toxic diffuse goiter, atypical toxic goiter, degenerating goiter and constitutional effects, tumors of the thyroid gland, goiters in unusual places, thyroiditis, hypothyroidism, hospital management, topographic anatomy and surgical technic. Hertzler begins with a premise that goiter is a lifetime disease, which, "once fully established," when persisting beyond adolescence or resisting medical treatment, does not afterward regress. His statements that the thyroid is "pretty much a useless organ and may be cast aside with advancing age" and that severe hypothyroidism, following complete removal, "occurs only in adolescents and not adults" are open to serious argument. True it is that a number of surgeons with extensive experience with goiter prefer a definite postoperative hypothyroidism to a mild hyperthyroidism, because of the danger of secondary surgery for residual hyperthyroidism, basing their contention on the fact that it is easier to feed thyroid than to dissect a definite scar for tiny thyroid remnants. Nevertheless, the present conception of adequate removal of toxic goiter is becoming more and more radical—that there is a tendency to leave less and less thyroid tissue after operation. Individual chapters devoted to nontoxic colloid goiter, goiter in childhood, toxic diffuse goiter and atypical toxic goiter are excellently presented as to pathology and symptomatology. One gets the feeling that the author has "surgical intentions" toward every goiter, colloid or otherwise, persisting beyond childhood. However, reading closely, one finds such a balancing statement as this: "The majority of operative failures come from trying to cure family rows by removing a simple colloid goiter." In proportion to the detailed thoroughness of the remainder of the book, the chapter on technic is rather thin. On the other hand, no one will ever learn thyroid surgery from a book or atlas. The uniform use of local anesthesia for toxic goiter is not a generally accepted procedure, even though in Hertzler's clinic "the surgeons here wear no mask, but they keep their mouths shut." In general the technic described and illustrated is that practiced by most goiter surgeons. Barring such criticisms as those here outlined, which after all represent merely a difference of opinion, this textbook represents a noteworthy contribution to surgery of the thyroid gland.

Précis de thérapeutique et de pharmacologie. Par A. Richaud et R. Hazard. Préface de A. Desgrez. Seventh edition revised by R. Hazard, professeur agrégé à la Faculté de médecine de Paris. Cloth. Price, 100 francs. Pp. 1,257, with 34 illustrations. Paris: Masson & Cie, 1935.

It is a pleasure to see a book on this subject come from the institution where François Magendie and Claude Bernard did so much for experimental pharmacology and medicine. Since their time, however, pharmacology in France has been in a great depression. Professor Richaud died some time ago and by his own wish the work of revision fell to Professor Hazard. In a short preface Professor Desgrez points out that since the appearance of the sixth edition ten years ago the advance in synthetic chemistry, chemical therapy, serotherapy, vaccine therapy, vitamins and general pharmacology makes such a revision necessary.

The task of renovating an old book, and especially one that has been successful, is difficult indeed. It is extremely difficult for a pharmacist to discard drugs that have long been and that are still prescribed, although critical work may have shown their use questionable or worthless. Consequently this book, while it adopts the new, hesitates to bury the dead. This is a universal failure in textbooks of pharmacology and therapeutics. As a "précis" it is quite large. The subject matter is divided into three parts: Part I is devoted to general pharmacology and occupies about one fourth of the whole. Part II is devoted to special pharmacology and occupies 845 pages. This is subdivided into (1) inorganic drugs, (2) synthetic organic chemicals, (3) products of vegetable origin, and (4) products of animal origin. Part III deals briefly with pharmacy, the prescription, dosage and allied subjects.

The general pharmacology is too brief to be considered adequate in our best medical schools. However, it is supplemented to a large extent in the special part, and the whole is satisfactory. There are no original tracings, but instead a few diagrammatic representations. This perhaps is not a drawback, since it is not the tracings per se but a knowledge of how they are obtained and individual work in obtaining them that is most valuable to the student. In a book of this size, one might expect some references to the original literature, but there are none. Instead, a few names are mentioned. The authors explain this omission on the ground that it often is difficult to determine the paternity of an experimental fact or discovery.

In part II the method of discussion may be illustrated by mentioning a few of the important drugs. The discussion of iodine occupies twenty pages, covering its preparation, characteristics, reactions, occurrence in nature, in water, in air, in sea water, in vegetables, in animals; under physiologic effects, its local action, general action and therapeutic applications and preparations. Ergot is disposed of in two pages. While digitalis gets nine, opium and its alkaloids occupy twenty-seven pages. Some of the work of the last two years is not mentioned; e. g., copper in hemoglobin formation or the excretion of morphine; but from a practical point of view the omissions are neither numerous nor serious.

Part III, which discusses the pharmacy and prescription writing, strikes one as being somewhat backward; modern prescription writing emphasizes simplicity; and, while Professor Hazard is in the main conservative, he gives an example of the complete prescription, which includes a base, an adjuvant, a corrective, a corrective adjuvant, an intermediate and an excipient. While he hastens to say that these are not always used, it is not sufficient. Such mixtures are unscientific, unnecessary and often detrimental. Worse still, they disorient and confuse the student, often beyond recovery in the time devoted to the subject. If they are mentioned at all, it should be to condemn them.

The book as a whole contains the essentials of pharmacology and therapeutics but is weak in pharmacology and contains an excessive number of drugs for therapeutics, many more than are needed. An excessive number paves the way to weakness in therapeutics. It is no condemnation to say that English

speaking people would gain little by a translation of this book. It can be recommended, however, to students and others who wish practice in reading French and who wish to get a view of French pharmacology and therapeutics at first hand.

Abnormal Arterial Tension. By Edward J. Stieglitz, M.S., M.D., F.A.C.P., Assistant Clinical Professor of Medicine, Rush Medical College of the University of Chicago. Edited by Morris Fishbein, M.D. Cloth. Price, $4. Pp. 261, with 66 illustrations. New York: National Medical Book Company, Inc.: Doubleday, Doran, 1935.

In this little volume the author succeeds in covering a good deal of ground, even if he does not go into considerable detail. He discusses the methods of taking blood pressure measurements and their symptoms. The factors that alter blood pressure within normal variations and cause elevation or depression are considered fairly well under various headings. It is interesting to note that in the discussion of the symptoms of hypertension the author classifies them as cardiac, neurologic and renal. He succeeds fairly well in giving an acceptable description of the subject. Coronary sclerosis he considers as part of chronic hypertension. Under the subject of acute hypotension the author has an opportunity to describe the condition of shock. The states of chronic hypotension, as Addison's disease and those resulting from disturbed nutritional states or chronic infections, are given some space. The subjects of prognosis and treatment are fairly good. There are many illustrations, charts, diagrams and reproductions of roentgenograms. This book can be recommended highly to the general practitioner and student.

Die Haut- und Geschlechtskrankheiten: Eine zusammenfassende Darstellung für die Praxis. Herausgegeben von Prof. Dr. Leopold Arzt und Prof. Dr. Karl Zieler. Lieferung 21/22. Band II. Hautkrankheiten entzündlicher Natur, I. Teil. Von Prof. Dr. Gabor Nobl. Hautkrankheiten entzündlicher Natur, II. Teil. Von. Prof. Dr. Oscar Nägell. Hautkrankheiten entzündlicher, vorwiegend beruflicher Natur und ihre Stigmata. Von Priv.-Doz. Dr. Alfred Perutz. Paper. Price, 16 marks. Pp. 235-540, with 173 illustrations. Berlin and Vienna: Urban & Schwarzenberg, 1935.

Die Haut- und Geschlechtskrankheiten: Eine zusammenfassende Darstellung für die Praxis. Herausgegeben von Prof. Dr. Leopold Arzt und Prof. Dr. Karl Zieler. Lieferung 23/24, Band II. Die Hypertrophien der Epidermis. Von Prof. Dr. M. Oppenheim. Die Hypertrophien des Pigmentes. Von. Prof. Dr. M. Oppenheim. Bindegewebshypertrophien, Pachydermien. Von Prof. Dr. Eduard Neuber. Atrophien. Von Priv.-Doz. Dr. Stefan Robert Brünauer. Neurosen der Haut. Von Priv.-Doz. Dr. Alfred Perutz. Paper. Price, 18 marks. Pp. 541-828, with 95 illustrations. Berlin and Vienna: Urban & Schwarzenberg, 1935.

In the volume devoted to a consideration of the inflammatory dermatoses are included chapters on the toxic erythemas, urticaria, prurigo, neurodermitis and the varicose symptom complex, a section on the papulosquamous dermatoses, and a thorough discussion of industrial dermatoses. Discussion of these subjects is detailed and well organized and is greatly aided by the liberal use of excellent reproductions of photographs in black and white and plates of moulages in colors.

The same thoroughness of discussion marks the volume devoted to a consideration of the hypertrophies and atrophies of the skin and pigment and to the cutaneous neuroses. The numerous illustrations tend to make these volumes a veritable atlas. While the books are obviously intended for the specialist in this field, the readability of the text and the excellence of the illustrations make them a valuable source of information for all physicians.

Dial Away Your Fat: Vest Pocket Calorie Calculator Does It! Paper. Price, 25 cents. Pp. 32. Los Angeles: The Health Builders, 1935.

This pocket-size booklet consists essentially of a series of alphabetically arranged and indexed tables of 100 calory portions of foods. It derives its name from a dial, attached to the front cover, with which the user is able to keep a tally of the number of calories eaten during the day. Space is provided in the back of the booklet for recording the daily total caloric intake and the daily weight. The tables are preceded by a brief discussion of the principles of weight reduction and a criticism of some commercially exploited nostrums. An initial diet of from 1,200 to 1,400 calories is advised, followed by an increase "if you are losing weight too rapidly" until "the weight loss is satisfactory." The booklet is an honest attempt to extract a profit from the obesity problem. The problem is properly presented as a balance between energy intake and energy expenditure and the information is sold as such and not as an inert powder or bath salt. The ready reference and recording features are commendable. However, this commercial venture is subject to the same serious objections as all similar projects. It assumes that the individual who attempts to use it will be normal except for overweight, and that he or she is capable of adjusting the diet both qualitatively and quantitatively so as to avoid the injurious effects of improper dieting. The experienced physician knows that these assumptions are not valid in a large percentage of cases. These booklets would have a much greater claim for recommendation if distributed only through physicians in conjunction with proper medical supervision.

King's and Some King's Men: Being a Record of The Medical Department of King's College, London, from 1830 to 1909, and of King's College Hospital Medical School from 1909 to 1934. By H. Willoughby Lyle, M.D., F.R.C.S., Fellow of King's College, London. Consulting Ophthalmic Surgeon to King's College Hospital. Cloth. Price, $9. Pp. 613, with 18 illustrations. New York & London: Oxford University Press, 1935.

This is a delightfully written account of the alma mater of many of the medical giants, past and present, in the British empire. To lovers of tradition this intimate narrative of steady progress, honest effort and scientific achievement should prove most instructive and illuminating. "Traditions," writes the author, "are made up chiefly of unwritten doctrines, customs, practices, and the endeavors of those who have 'gone before' and are transmitted from one generation to another. Traditions are in all probability the most valuable assets which a medical school and hospital possess, and they impart to it a personality which goes to make both respected and even beloved." In the United States the Massachusetts General Hospital, Boston, can point to such love and veneration for tradition. Lord Lister, Watson-Cheyne, Lenthal Cheatle, Thomas Percy Legg and Hutchinson Roy Bell are names familiar to all who can truly call themselves surgeons in the United States. The list of internists and other specialists is equally imposing. Many amusing incidents and anecdotes of more than local interest are recounted, among them the uncalled for antagonisms on the part of members of the nursing staff who objected to Lord Lister's operating on a young boy for acute osteomyelitis of the leg and sought forcibly to prevent the patient's being carried from the ward to the operating room. Also significant is the fact that Lister would accept the chair of surgery at King's only on condition that four men whom he had trained in Edinburgh be allowed to come with him. One learns that Cheyne became a surgeon-rear-admiral during the World War, that hundreds of King's graduates served their country, fifteen of them being killed in action. Of purely "alma mater" interest are the histories of the various King's clubs and societies, from cricket to tennis; but their achievements make one realize how close a corporation was this combination of school and hospital. A list of the teaching staffs, medical associates, scholarships and prizes won by King's men and an excellent index complete the volume. One of the illustrations shows King's College Hospital as it was in the year 1839.

Diseases of the Nervous System: A Text-Book of Neurology and Psychiatry. By Smith Ely Jelliffe, M.D., Ph.D., and William A. White, M.D., Superintendent of St. Elizabeth's Hospital, Washington, D. C. Sixth edition. Cloth. Price, $9.50. Pp. 1,175, with 510 illustrations. Philadelphia: Lea & Febiger, 1935.

This is the most modern and thorough textbook of its kind. In addition to having all the good qualities of the preceding edition, the present one may rightly boast of containing descriptions of the most recent methods and tests and discussions of present-day thought and investigations in relation to neuropsychiatry and general medicine. The specific changes made in addition to the careful revision of the entire work are the addition of new methods of examination, recent metabolic studies in relation to the pathways of the hypothalamus, surgery of the sympathetic nervous system, an entirely new chapter on the endocrine entities, revision of the chapter on spinal cord lesions, addition of some of the less common peduncular and midbrain lesions, new thoughts in cerebral neoplastic localization, and considerable addition of new material to the psychic or symbolic systems with explanatory interpretation of what one finds in these various psychic entities. There is a good bibliography, and the illustrations are excellent. The text and the printed type are easy to read. This book is highly recommended for use by students, practitioners and neuropsychiatrists.

Commoner Diseases of the Skin. By S. William Becker, M.S., M.D., Associate Professor of Dermatology, School of Medicine, University of Chicago. Edited by Morris Fishbein, M.D. Cloth. Price, $4. Pp. 283, with 83 illustrations. New York: National Medical Book Company, Inc.: Doubleday, Doran & Co., 1935.

This book presents, in lecture style, full discussions of the more common skin diseases. Underlying principles are emphasized so that, while not encyclopedic in the sense of a textbook, it covers the field of general dermatology fairly well. The physiologic and biologic approach is used in describing the etiology. The field of allergy as related to the skin is well covered and up to date. The chapters on the neurodermatoses and pigmentary dermatoses of the skin are especially good. In treating neurodermatoses, the author has stressed the necessity of treating the nervous exhaustion that occurs so frequently in patients suffering from this disease. The treatment of certain forms of dermatoses are illustrated by case histories. The photographic illustrations are excellent and well selected in connection with the text. The author has limited the therapy to one or two well chosen and accepted forms of treatment for each disease. There is also a separate chapter devoted to pharmaceutical details and prescriptions. The value of psychotherapeutic measures in dermatology is discussed. The volume covers the field of dermatology adequately for the general practitioner and student.

Medicolegal

Workmen's Compensation Acts: Physician's Right to Initiate Proceedings Before Industrial Board.—Medical treatment to an injured employee was rendered by Dr. Meherin, on authorization of the employer. Later, being unable to collect his fees, Dr. Meherin filed an original application with the industrial accident commission of California, naming himself as applicant and the injured employee and his employer and its insurance carrier as defendants and asking an adjustment of his claim. A hearing was had on the application, at which the employee did not appear. Subsequently, at the invitation of the referee, he joined in the proceedings and submitted his claim on the record as it then stood. Thereupon the commission awarded him compensation and included in it an award for medical treatment, payable directly to Dr. Meherin. The state insurance commissioner, as liquidator of the insurance carrier, petitioned the Supreme Court of California for a review and annulment of the award.

The chief objection to the award was based on the facts that the original application for adjustment of the claim was filed not by the injured employee but by Dr. Meherin, and that within the time limited by the statute no proper application was filed by either the employer or the employee. There can be no question, said the Supreme Court, that if Dr. Meherin was not entitled to file the application, no proper application was timely filed and the whole award must be annulled. On the other hand, if Dr. Meherin was entitled to file the original application, the employee could be brought in by the industrial accident commission at any time during which the commission had continuing jurisdiction and was entitled to the same consideration as if he had initiated the proceedings. The physician in this case named as defendants all parties having any possible interest in the subject matter. In effect, he filed an application not alone for himself but also on behalf of the injured employee. The basis of the proceeding was not an alleged independent right of the physician against the employer but the liability of the employer to the employee, to which the incidental liability of the employer to the physician was a necessary concomitant. If the employee was properly a party to the proceeding, said the court, no difficulty arose with reference to making an award in his favor and impressing thereon a lien in favor of the physician.

The workmen's compensation act of California authorizes the filing of an application "by any party in interest." Although a physician's right to payment for medical services is incidental to the employee's right to compensation, concluded the court, nevertheless a physician who has rendered medical services to an injured employee has an interest in the subject matter of the controversy sufficient to bring him within the class designated by the act as "parties in interest" who may file an application. The award of the industrial commission was consequently affirmed.—*Independence Indemnity Co. v. Industrial Accident Commission (Calif.), 41 P. (2d) 320; Same, 41 P. (2d) 327.*

Malpractice: Needle Left in Abdomen.—Sugaya was referred by his attending physician to the physician-defendant for an appendectomy. At operation the appendix was found to be perforated and was removed. The incision was closed, except where provision was made for drainage. The physician-defendant attended the patient during the critical period following the operation. Thereafter the attending physician managed the case and made the necessary changes in the surgical dressings. About three and one-half months after the appendectomy a straight needle was located in the abdomen by roentgenograms and was surgically removed from the psoas muscle by a third physician. The patient then sued the physician-defendant. From a judgment of the trial court in favor of the patient, the physician-defendant appealed to the district court of appeal, first district, division 2, California.

The only question to be decided, said the district court of appeal, is whether the indirect evidence, in the absence of direct evidence, was sufficient proof that the needle removed from the patient's psoas muscle entered the patient's abdomen during the appendectomy. A medical witness testified that the needle might have arrived at the location from which it was removed (1) through swallowing, (2) during the operation or (3) during the after-treatment by the attending physician. The patient testified that to his knowledge he had not swallowed the needle nor had he had a previous operation. The attending physician testified that he used no needle in the treatment after the operation. And the physician-defendant testified that, while straight needles were carried on the nurse's tray during the operation, he himself had used only curved needles. Assuming this testimony to be true, said the court, then the needle must have arrived in the patient's abdomen through inadvertence and without any actual knowledge on the part of any one. The only time when needles similar to the one described in the evidence were in close proximity to the patient's abdomen was during the operation. The jury could properly conclude, said the court, that it was more probable that the needle entered the abdomen through inadvertence during the operation and while the wound was fully open than at any time before or after. The judgment of the trial court in favor of the patient was affirmed.—*Sugaya v. Morton (Calif.), 40 P. (2d) 581.*

Typhoid Fever: Liability of Municipality for Typhoid Fever Contracted from Municipal Water Supply.—The defendant city of Helena, a municipal corporation, owned and operated a water supply system from which it furnished water for drinking and domestic purposes on a rental basis. A sewer line installed by the city passed above the Eureka water main through which the city furnished water to the plaintiff. As early as July depressions in the street were noticed over the site where the water main and the sewer crossed. On September 30 the city discovered a break in the sewer line within a few feet of the point of crossing. A fluorescein and salt test to determine whether sewage was entering the water main was negative. Samples of water taken between August 6 and August 20, however, were reported by the state board of health as showing the presence of colon bacilli. On August 20 the first case of typhoid fever was discovered, but seven days later the mayor and the city health officer assured the inhabitants of the city, through the medium of a newspaper, that the water supply was safe and that it was unnecessary to boil drinking water. On or about September 15 the plaintiff, a consumer of water from the city's Eureka water supply system, showed symptoms of typhoid fever. Chlorination was begun on September 14 or 16 and continued until September 26, when the Eureka water supply was discontinued. Following this the outbreak of typhoid fever was promptly controlled. The plaintiff sued the city and from a judgment in his favor the city appealed to the Supreme Court of Montana.

That the water supplied by the city contained typhoid fever germs, said the Supreme Court, is fairly inferable from the facts that after the sewer was repaired the water was shown by tests to be pure and that after the water was chlorinated

there was a sharp decrease in the number of typhoid fever cases and shortly thereafter no more cases developed. The evidence supported a finding by the jury that the city had failed to use reasonable care to safeguard the purity of its water supply. The city cannot furnish water to its inhabitants which it knew, or in the exercise of reasonable care should have known, was polluted with sewage escaping from a defective sewer pipe, without assuming liability for damages occasioned thereby.

Judgment in favor of the plaintiff was affirmed.—*Safransky v. City of Helena (Mont.), 39 P. (2d) 644.*

Society Proceedings

COMING MEETINGS

American Academy of Tropical Medicine, St. Louis, Nov. 20-21. Dr. Earl B. McKinley, 1335 H Street N.W., Washington, D. C., Secretary.
American Association of Railway Surgeons, Chicago, November 13-15. Dr. Louis J. Mitchell, 86 E. Randolph St., Chicago, Secretary.
American College of Surgeons, San Francisco, October 28-November 1. Dr. George W. Crile, 40 East Erie St., Chicago.
American Society of Tropical Medicine, St. Louis, November 19-22. Dr. Alfred C. Reed, 350 Post Street, San Francisco, Secretary.
Association of American Medical Colleges, Toronto, Canada, Oct. 28-30. Dr. Fred C. Zapffe, 5 South Wabash Avenue, Chicago, Secretary.
Central Society for Clinical Research, Chicago, Nov. 1-2. Dr. Lawrence D. Thompson, 3720 Washington Boulevard, St. Louis, Secretary.
Clinical Orthopedic Society, Indianapolis and Louisville, Nov. 15-16. Dr. J. E. M. Thomson, 1307 N Street, Lincoln, Neb., Secretary.
Medical and Surgical Association of the Southwest, El Paso, Texas, Nov. 21-23. Dr. W. Warner Watkins, 15 East Monroe Street, Phoenix, Ariz., Secretary.
National Society for the Prevention of Blindness, New York, Dec. 5-7. Dr. Lewis H. Carris, 50 West 50th Street, New York, Managing Director.
New York State Association of Public Health Laboratories, Albany, Nov. 1. Miss Mary B. Kirkbride, New Scotland Avenue, Albany, Secretary.
Omaha Mid-West Clinical Society, Omaha, Oct. 28-Nov. 1. Dr. J. D. McCarthy, 107 South 17th Street, Omaha, Secretary.
Pacific Coast Society of Obstetrics and Gynecology, Los Angeles, Nov. 6-9. Dr. T. Floyd Bell, 400 29th Street, Oakland, Calif., Secretary.
Radiological Society of North America, Detroit, Dec. 2-6. Dr. Donald S. Childs, 607 Medical Arts Building, Syracuse, N. Y., Secretary.
Society of American Bacteriologists, New York, Dec. 26-28. Dr. I. L. Baldwin, College of Agriculture, University of Wisconsin, Madison, Wis., Secretary.
Southern Medical Association, St. Louis, November 19-22. Mr. C. P. Loranz, Empire Building, Birmingham, Ala., Secretary.
Southern Surgical Association, Hot Springs, Va., Dec. 10-12. Dr. E. W. Alton Ochsner, 1430 Tulane Ave., New Orleans, Secretary.
Western Surgical Association, Rochester, Minn., Dec. 6-8. Dr. Albert H. Montgomery, 122 South Michigan Boulevard, Chicago, Secretary.

AMERICAN ASSOCIATION FOR THE STUDY AND CONTROL OF RHEUMATIC DISEASES

Fourth Conference on Rheumatic Diseases

Report of Proceedings at Atlantic City, N. J., June 10, 1935

In the last fifteen years there has been a marked increase in the interest taken by physicians in the care of patients with arthritis. Fifteen years ago there were perhaps twenty American "rheumatism specialists." Now there are more than a hundred physicians in this country aside from spa physicians whose major concern is the arthritides. The American Committee for the Control of Rheumatism has had a large share in stimulating this interest by holding demonstrations and conferences at the time of annual sessions of the American Medical Association. Two years ago the increasingly large unorganized audience took matters into its own hands and formed the American Association for the Study and Control of Rheumatic Diseases. Last June, at Atlantic City, the Fourth Rheumatism Conference was held on the occasion of the second meeting of the association. The modest room, of a size previously quite ample, was completely filled, many standing even in the adjacent hall. In eight hours nineteen papers and seventy-six discussions were presented.

The conception of chronic arthritis as a general and not a local disease is gaining ground. Therefore, Dr. Ernest E. Irons of Chicago, president of the association, emphasized that studies on arthritis must be diverse and include research not only on the joints but on all body tissues and functions in order to envision the patient as a whole and to study the soil in which arthritis thrives. Although the majority of arthritic patients must be cared for by general practitioners, it may eventually be necessary for the proper study of arthritis to establish hospitals where research may be highly developed by investigators qualified in its medical, orthopedic, bacterial, chemical and metabolic aspects. A few such clinics already exist at certain medical centers. Dr. Irons felt that they will be more successful if developed as part of a large general clinic. Clinics for the treatment of arthritis alone, established apart from other medical projects, may offer superior custodial advantages for patients needing prolonged hospitalization, but such clinics, Dr. Irons thought, are likely to suffer from lack of stimulation from other departments of a general clinic, and the relation of arthritis to medicine as a whole might be jeopardized.

The division of chronic arthritis into two great common types, atrophic (infectious, proliferative, rheumatoid) and hypertrophic (senescent, degenerative, osteo-) arthritis, is serviceable. There is still disagreement as to whether they represent separate entities. Although the majority believe they are, Irons reiterated the suggestion that each may not have one different specific cause but that an articular disease may assume this or that course, with the production of one or another type of arthritis by reason of the kind, age and resistance of affected tissues rather than the nature of the initial insult.

Analyzing forty-nine roentgenologic characteristics, Ferguson, Kasabach and Taylor of New York concluded that, although varieties of bone atrophy, lipping and zones of erosion are common to the roentgenograms of different types of arthritis (atrophic, hypertrophic, gouty, gonorrheal, and others), each type has its characteristic picture or combination of roentgenographic alterations, which are best seen in roentgenograms of the hands, feet and knees. Key of St. Louis insisted that the clinical observations are more important in diagnosis than roentgenograms, and that a clinical diagnosis cannot be ventured by the roentgenologist unless he knows the approximate duration of the arthritis and whether the part has been in use. Otherwise, an interpretation of the probable type of atrophy of bone, for example, is impossible. Ferguson agreed that this was true and that in some cases of acute gonorrheal arthritis of short duration the roentgenogram may resemble that of old tuberculous arthritis, a differentiation possible only if the duration of the disease is known to the roentgenologist. Most roentgenologists who see any overgrowth of bone in a case of articular disease make a diagnosis of "hypertrophic arthritis," and if overgrowth is absent the diagnosis becomes "atrophic arthritis." Pemberton of Philadelphia felt that radiologists must exhibit a more informed type of scrutiny and must interest themselves in the variable clinical and pathologic aspects of the disease in order that roentgenograms might be interpreted more correctly.

Atrophic (Infectious, Proliferative, Rheumatoid) Arthritis

Organisms, generally green-producing streptococci but occasionally hemolytic streptococci, were found in the blood of 6 per cent of controls and in that of 21 per cent of patients with atrophic arthritis by McEwen, Bunim and Alexander of New York. Agglutinins and precipitins for hemolytic streptococci were found in the blood of 88 and 80 per cent respectively of thirty-seven patients with atrophic arthritis. Precipitins were also found frequently in other types of arthritis, even gonorrheal. Antistreptolysins and antifibrinolysins were found only occasionally by McEwen, but as Dawson of New York said these tests are indexes of recent, acute hemolytic streptococcus infection and one would not expect to find them in cases of chronic atrophic arthritis, although they are sometimes found in early acute cases. Cecil of New York felt that these observations favored the infectious theory of the disease and the streptococci so isolated are not contaminants, although it is not yet known how they get into the blood stream. The bacteriologic terminology of the different workers must not lead to confusion, for many streptococci undergo mutation and what one investigator calls Streptococcus viridans

may under varying conditions become slightly or frankly hemolytic. Whether a high or low percentage of positive blood cultures and agglutination tests are obtained depends on the selection of cases of atrophic arthritis, whether they are acutely active or smolderingly chronic. Key has been unable to isolate streptococci from articular tissues but finds staphylococci in about a third of the joints so studied.

In cases of atrophic arthritis the sedimentation rate of erythrocytes is generally rapid, and in cases of hypertrophic arthritis it is generally normal or but slightly elevated. The rate of sedimentation is believed to depend largely on the concentration of plasma fibrinogen and globulin and on cell volume. To determine the reasons for altered sedimentation rates in cases of atrophic arthritis, Davis of New York studied the various blood proteins. The total protein content of the blood is normal in both atrophic and hypertrophic arthritis, but in atrophic arthritis there is an increase in the plasma fibrinogen, a rise in the globulin fraction, and a fall in the albumin fraction. The greatest increase is in the euglobulin fraction. The albumin-globulin ratio is frequently less than 1. As the patient recovers, the ratio tends to return to normal. The estimation of the sedimentation rate is therefore a simple way of roughly detecting any alteration in the globulin or fibrinogen content of the blood, provided a correction is made for cell volume. These changes are not found in cases of hypertrophic arthritis and they suggest again that atrophic and hypertrophic arthritis are quite different diseases. The rise in plasma globulin suggested to Davis that atrophic arthritis is an infectious disease, as rises often occur in such diseases. The fall in albumin suggests a factor of malnutrition or vitamin deficiency, or both. In cases of atrophic arthritis, if the plasma albumin is quite low, considerable edema may appear. Restriction of protein may thus be harmful and a generous intake of protein may lessen edema in such cases.

Discussing the possible significance of these alterations, Haden of Cleveland said that he had found somewhat similar changes in cases of hyperthyroidism, a disease presumably not infectious. These observations complete the cycle of reported or reputed metabolic abnormalities in cases of atrophic arthritis. Physicians have long been warned of alterations in the utilization of carbohydrates, apparently present in many cases of atrophic arthritis. At last year's conference, alterations in blood fats were stressed, and now alterations in protein fractions. Since the metabolism of purines, several minerals and vitamins have previously been incriminated, one is now at liberty to suspect abnormalities in the whole metabolic spectrum. In any event these interesting observations illustrate how profound and widespread are the disturbances that constitute the syndrome of this disease.

TREATMENT

The value of a generous protein diet in cases of atrophic arthritis with edema has been mentioned. A diet low in carbohydrate has long been considered by some to be the standard diet for patients with atrophic arthritis. To determine whether excess carbohydrate is harmful for such patients, Bowen and Lockie of Buffalo included about 500 Gm. of carbohydrate in the diet of eight patients who were observed for from fifteen to sixty-five weeks. The nutrition of the majority improved steadily and they gained from 24 to 50 pounds (11 to 23 Kg.). The arthritis was not materially affected, and in general no exacerbations were noted. Insulin given to some did not increase the appetite or augment the effect of the diet. No changes in respiratory metabolism were noted. Some workers in discussion interpreted these remarks as the launching of a new diet for the already overdieted arthritic patient. They merely indicated that a fairly generous supply of carbohydrate was obviously not harmful to the arthritic patient and that Bowen and Lockie felt that restrictions in carbohydrate were of no particular merit either. It was mentioned that in patients whose atrophic arthritis was temporarily inactivated completely by intercurrent jaundice the administration of from 400 to 600 Gm. of carbohydrate daily for many weeks did not counteract the analgesic effect of jaundice or bring out symptoms of arthritis.

Bauer of Boston was doubtful that vitamin deficiency plays an important part in the production of atrophic arthritis. No different results were obtained by a group of his patients who for from three to five years adhered to a special high vitamin diet than were obtained by a group who did not.

Hartung of New York believed that the hematopoietic and leukopoietic functions of the body do not respond properly in the presence of the toxemia or infection causing atrophic arthritis. The infection may be so low in virulence that bone marrow is not stimulated, or there may be a fundamental hormone deficiency somewhat like that in pernicious anemia. Since nucleic acid, one of the disintegration products of leukocytes, is known to stimulate the myelopoietic function of bone marrow, Hartung decided to administer a concentrated extract of leukocytes intragluteally to ten patients daily for long periods. Six patients showed prompt symptomatic and constitutional improvement. There was, however, no change in the appearance of the blood cells or in the sedimentation rate. The effect was not specific and may have resulted from a nonspecific protein reaction, but it was obtained more quickly, was more lasting, and was superior to that obtained by him with any other foreign protein.

Snyder of New York and his colleagues were of the opinion that cinchophen, a very useful drug, has been condemned unjustly. Reviewing all reported cases of cinchophen toxicity, they concluded that in only a few was cinchophen unmistakably responsible. After having administered cinchophen to 2,560 patients in ten years, they observed but one case of catarrhal jaundice. Urticaria developed in a few cases but severe jaundice or atrophy of the liver in none.

Physical therapy is perhaps the most helpful form of treatment, but it is used inadequately. Coulter of Chicago was of the opinion that, to supplement the limited sessions of professional physical therapy which patients could afford, the intelligent ones and their relatives should be fully instructed in simple methods of physical therapy that could be applied in the home. Thus the cost of the patient's care could be reduced and the physician could control the patient over the long periods necessary for treatment. By demonstrations and mimeographed instructions Coulter teaches his patients the optimal types and amounts of heat, massage and exercise, the use of cheap but effective electric "bakers," whirlpool baths, and a simple machine for the production of sinusoidal-faradic current, and various appliances for specific, harmless exercises. In Massachusetts alone there are about twenty-two arthritic patients for every physician, and according to Ober of Boston about 70,000 patients receive no treatment whatever. But before home physical therapy can be safely prescribed physicians themselves must learn its principles and methods. A visiting team of physicians, nurses and physical therapists skilled in the management of arthritis should go among local physicians. Ober felt that only under the supervision of the local physician should a visiting physical therapist teach and prescribe home physical therapy. He mentioned that 75 per cent of his arthritic patients have previously sought the services of cultists, who gave them cheap, sometimes pain-relieving treatments for which, generally, no substitute had been given by the physician. Unless the better forms of professional and home physical therapy are utilized more fully, patients will continue to flock to the spine and foot twisters.

After summarizing the results from various parts of the country, Ober found that fever therapy, which has proved so helpful in cases of gonorrheal arthritis, does not "cure" chronic atrophic arthritis. Of a total of 315 patients so far treated in different clinics, only 5 per cent became symptom free. Twenty-five per cent were notably relieved. The remainder received little or no benefit. Results are somewhat better in cases of acute atrophic arthritis: Of twenty-one patients so treated, 10 per cent seemed completely relieved, 40 per cent notably helped. Stainsby of New York and Bauer of Boston agreed that remissions are occasionally induced but that fever therapy should not be used to the exclusion of other measures.

Simpson of Dayton, Ohio, and Shands of Durham, N. C., felt that fever therapy given just before or after orthopedic manipulations augmented the benefits of the latter. The recognition of impending deformities is the responsibility of the attending physician who, according to Swaim of Boston, should early seek the full cooperation of an orthopedic colleague.

Hypertrophic (Senescent, Degenerative, Osteo-) Arthritis

From the roentgenologic point of view, as Haden pointed out, there are several types of "hypertrophic arthritis," including that seen in traumatized joints and in certain stages of gouty, gonorrheal, atrophic and other forms of arthritis. "Primary hypertrophic arthritis" represents an entity and results from degeneration of joint cartilage, with proliferation of subchondral bone, incident to physiologic aging and the wear and tear of continued use. Analyzing fifty such cases to determine the accelerating factors of the disease, Haden concluded that infection is a minimal factor and that obesity and an altered metabolic rate (low in 84 per cent of cases) are the common provocatives, the correction or prevention of which may do much to lessen the incidence and disability of hypertrophic arthritis. Bauer and Bennett studied the knee joints of persons from the first to the ninth decade of life. Beyond the second decade the degenerative changes that lead to hypertrophic arthritis were found with increasing frequency, so that by the fifth decade all knee joints were so affected. Some persons inherit better cartilage than others. The poorer the inheritance and the greater the endogenous and exogenous trauma to which the cartilages are subjected, the earlier will hypertrophic arthritis develop. Davis, as noted, found little or no changes in the sedimentation rate or in blood proteins in hypertrophic arthritis, and Dawson found no agglutinins to hemolytic streptococci. In a discussion on fever therapy it was stated that patients with hypertrophic arthritis were not particularly benefited by it.

Gonorrheal Arthritis

The gonorrheal fixation test is of great aid in confirming a clinical diagnosis of gonorrheal arthritis, according to McEwen and his colleagues. It was positive in 98 per cent of forty-three cases. Dawson also felt that the test is of great value, but Key warned that in a case of chronic nongonorrheal arthritis associated incidentally with acute gonorrheal urethritis a positive complement fixation test might lead to error regarding the etiology of the arthritis.

In a summary to the conference on the results to date in various parts of the country of fever therapy in the treatment of gonorrheal arthritis it was noted that 80 per cent of 151 patients with acute gonorrheal arthritis have apparently been cured and that an additional 10 per cent have been markedly relieved. The results in chronic gonorrheal arthritis (of more than six weeks' duration) are less striking but also good. Of thirty-two such patients treated, 35 per cent were cured and 30 per cent markedly relieved. The joints are apparently sterilized rapidly by the bacteriolytic effect of several hours of fever at 106 to 107 F. Bauer and Simpson both reported excellent results and considered fever therapy the treatment of choice and almost specific for gonorrheal arthritis. No untoward reactions were reported except by Snyder, who noted progressive, uncontrollable and fatal hyperpyrexia induced by fever therapy in one case of gonorrheal arthritis.

Rheumatic Fever

A review by Nichol of Miami of the available data again indicates that there is much less rheumatic fever in southern than in northern states. During the past five years, for example, the admission rate of patients with rheumatic fever, rheumatic carditis and chorea in a general hospital in Miami was only a tenth of what it was in Boston. Levine of Boston stated that in New Orleans the incidence of mitral stenosis was a twentieth of that in Boston, and he concluded that there is either much less rheumatic fever in the South or that it does not affect the heart as it does in the North.

Of 342 rheumatic children observed by Shapiro of Minneapolis over a period of eight years, 52 per cent had only one attack; 48 per cent had recurrences, generally within the first four years after the initial attack. This is probably the normal expectancy of recurrences. The majority of "growing pains" of children are not rheumatic. Shapiro distinguished the latter from rheumatic growing pains as follows: The "nonrheumatic variety" are usually diffusely and vaguely located in the legs, appear or are worse at night, are not associated with fever, and produce no limping or significant stiffness during the day. The "rheumatic variety" are less diffuse, are more commonly articular, are generally better at night if the patient is warm, and worse in the daytime and on walking, particularly during the first hour of the day, and are often associated with a little fever, articular swelling, and heat. Children with growing pains of the latter variety are in danger of rheumatic carditis.

Swift of New York reviewed current ideas on the nature of rheumatic fever. The tissues of patients with this disease apparently are unusually vulnerable to certain injurious agents, especially streptococci or their derivatives. Although it has not been proved that streptococci or a virus are the cause of the disease, the rheumatic patient must be protected, particularly from streptococcic infections, in order to prevent or diminish relapses. McEwen found streptococci, chiefly Streptococcus viridans, in the blood of 24 per cent of patients with rheumatic fever and in 6 per cent of controls; agglutinins to hemolytic streptococci were found only occasionally. Precipitin tests were positive in 50 per cent of cases, and antistreptolysis and antifibrinolysin titers were generally, though not always, high.

Rinehart of San Francisco advanced the idea that rheumatic fever may be the result of the combined influence of deficiency in vitamin C and infection. He reported that in guinea-pigs on a diet devoid of vitamin C, which were inoculated with a guinea-pig strain of hemolytic streptococci or other organisms, cardiac and articular lesions developed with considerable frequency, notably resembling those of rheumatic fever. Guinea-pigs infected with these organisms but on an adequate diet did not show such lesions, and those treated with a deficient diet alone showed only slight lesions. In the discussion of this paper, doubts were expressed as to whether the articular lesions were rheumatic or scorbutic. Swift agreed that lesions of some sort were thus experimentally produced, but he stated that clinical proof was lacking that there was any difference in the metabolism of vitamin C between rheumatic and nonrheumatic patients. Patients with the active and quiescent rheumatic fever were treated with cevitamic acid. Patients with the active form were not benefited and those with the quiescent form later had the usual number of recurrences.

TREATMENT

Kaiser of Rochester, N. Y., urged tonsillectomy for every rheumatic child. According to his large statistics the child who has had his tonsils out is less likely to have rheumatic manifestations and, if he does have them, serious cardiac complications are less likely to develop. Although the incidence of recurrent attacks is not influenced by tonsillectomy, the mortality among 600 rheumatic children was about twice as high as for those who had tonsils at the time of the initial attack as for those whose tonsils had been removed. Kaiser stated that tonsillectomy, however, does not influence the incidence of chorea or muscular rheumatism. The most definite indication for tonsillectomy in children is a history of repeated attacks of tonsillitis.

Vaccine seemed of little value to Stroud of Philadelphia. As many recurrences occurred among his patients who were treated with vaccine as among a control group.

Chorea

Fever reactions to typhoid vaccine given daily for several days were found by Sutton and Dodge of New York to shorten attacks of chorea an average of thirty-six days, as compared with results when physical therapy, drugs and diets were used. The average duration of the attacks of 150 patients treated by vaccine reactions was 8.5 days; that of 150 patients treated otherwise, 42.6 days. By means of one or two sessions of fever therapy at 105 to 106 F. for five hours, results as good as with vaccine were obtained in sixteen cases. Fever therapy is preferable, as foreign protein shock is avoided, the fever is controllable, and only two sessions instead of several daily reactions are necessary. An associated, active rheumatic carditis does not interfere with such therapy. Indeed, in several cases in which such treatment was employed signs of carditis diminished. It was noted that Sutton and Dodge's results with fever therapy in the treatment of chorea are similar to those reported in twenty-five cases at the Fifth Fever Therapy Conference and in ten cases at the Mayo Clinic.

Current Medical Literature

AMERICAN

The Association library lends periodicals to Fellows of the Association and to individual subscribers to THE JOURNAL in continental United States and Canada for a period of three days. Periodicals are available from 1925 to date. Requests for issues of earlier date cannot be filled. Requests should be accompanied by stamps to cover postage (6 cents if one and 12 cents if two periodicals are requested). Periodicals published by the American Medical Association are not available for lending but may be supplied on purchase order. Reprints as a rule are the property of authors and can be obtained for permanent possession only from them.

Titles marked with an asterisk (*) are abstracted below.

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Permanent Type of Ditch Construction. A. H. Fletchner, Memphis, Tenn.—p. 897.

Measles, Scarlet Fever and Whooping Cough in Los Angeles County Health Department Area: Ten Year Study. H. O. Swartout, Los Angeles.—p. 907.

Teaching of Epidemiology by Applicatory Problems. E. L. Munson, San Francisco.—p. 913.

*Eosin Methylene Blue Agar for Rapid Direct Count of Endamoeba Coli. H. W. Gehm and H. Heukelekian, New Brunswick, N. J.—p. 920.

Diseases of Peasants of Haiti. C. Lhérisson.—p. 924.

Sewage Contaminated Irrigation Water: Major Public Health Program in West. E. N. Chapman, Colorado Springs, Colo.—p. 930.

Outbreak of Food Poisoning, Probably Due to Staphylococcus Aureus. A. Corpening and Elsie P. Foxhall, Richmond, Va.—p. 938.

Development of Adult Type Pulmonary Tuberculosis Following Recognition of Childhood Form. H. R. Edwards, New Haven, Conn.—p. 941.

Bacteria on Fresh Fruit. Marion M. Johnston and Mildred J. Kaake, Toronto.—p. 945.

Diphtheria in Grays Harbor County, Washington. Ruth R. Laue, Aberdeen, Wash.—p. 948.

Flipping Device for Flange Rubber Stoppers. V. T. Schuhardt and J. H. Brewer, Austin, Texas.—p. 951.

Neglected Opportunity for Control of Respiratory Disease. H. N. Calver, New York.—p. 953.

Eosin Methylene Blue Agar for Count of Endamoeba Coli.—Gehm and Huekelekian compared eosin methylene blue agar with and without crystal violet (1:100,000) and found that, although the presence of crystal violet cuts down the contaminants, it also cuts the colon numbers to an extent that would prohibit its use as a direct counting medium. Eosin methylene blue agar is prepared according to the standard method, except that the use of 2 per cent rather than the usual 1.5 per cent of agar gives a firmer surface for smearing. Plates are poured to a depth of about 3 mm. One cc. of the inoculum is run on the surface of the medium and smeared with the pipet so that the entire surface is covered. The plate is then tilted back and forth several times so that a more even distribution is obtained. The plate is placed in a 37 C. incubator and the cover is removed. Drying of the inoculum occurs in about one hour. To obtain perfect distribution it is necessary to tilt the plate, as done at the start, when the plate is about half dry. When the surface is thoroughly dry, the plate is closed, inverted and incubated twenty-four hours, and characteristic colonies are counted. Contamination from leaving the agar surface exposed in the incubator was practically nil in incubators that were not particularly clean. The dyes present in the medium evidently inhibit bacteria that may be deposited on the agar surface with the dust from the air. Plates containing between thirty and 100 Endamoeba coli colonies give the most consistent counts. As the authors were interested in applying this method to sewage in various stages of treatment and heavily polluted waters, comparative counts were made against the brilliant green bile broth count. The results show a close agreement of the counts by the two methods. In thirteen cases out of nineteen the counts by the eosin methylene blue agar method were higher than those by brilliant green broth bile. They found the method particularly adaptable to sewerage plant control and sewage research. Application of this method to river waters not heavily polluted appears possible. In this work a comparison should be made with lactose broth with eosin methylene blue confirmation to see whether the inaccurate, cumbersome enrichment count may be omitted entirely.

Archives of Dermatology and Syphilology, Chicago

32: 363-544 (Sept.) 1935

Systematized Amyloidosis of Skin and Muscles. H. E. Michelson, Minneapolis, and F. W. Lynch, St. Paul.—p. 363.

Pachyonychia Congenita: Report of Case. A. W. Sohrweide, Syracuse, N. Y.—p. 370.

*Xanthoma Tuberosum: Report of Familial Occurrence with Probable Cardiac Lesions. C. G. Lane, Boston, and J. Goodman Jr., Baltimore.—p. 377.

Clinical Spectroscopy: Advantages and Physical Principles of Spectrograph and Technic in Taking Specimen for Biospectrometric Analysis. L. E. Gaul and A. H. Staud, New York.—p. 385.

LXXIII. State and Localization of Inorganic Salts in Skin as Revealed by Extraction and Micro-Incineration. D. J. Kooyman, St. Louis.—p. 394.

*Rôle of Iodine in Therapy of Syphilis: Discussion of Its Relationship to Lipoids. E. T. Burke, Salford, England.—p. 404.

*Dermatophytosis: Its Treatment with Trichophytin. E. F. Traub and J. A. Tolmach, New York.—p. 413.

Treatment of Arsenical Hepatitis with Sodium Dehydrocholate: Experimental and Clinical Studies in Cases of Arsphenamine Poisoning. B. Appel and I. R. Jankelson, Boston.—p. 422.

Epidemiology of Yaws. C. S. Butler, Brooklyn.—p. 446.

Evolution of Atopic Dermatitis. L. W. Hill, Boston, and M. R. Sulzberger, New York.—p. 451.

Acid in Blood as Cause of Diseases of Skin. J. E. Ginsberg, Chicago.—p. 464.

Familial Occurrence of Xanthoma Tuberosum.—Lane and Goodman report two cases of generalized xanthomatosis in a father and daughter, both with cardiac lesions, one with a history of an acute rheumatic attack and the other with a history of repeated sore throat and tonsillitis. Rheumatic scarring may result in secondary xanthomatous deposits on the endocardium, just as trauma results in deposits on the skin. A third case is reported, occurring in a child in the same family, with an elevated value for serum cholesterol but without cutaneous lesions. This child may have had the underlying fault in metabolism, but the various lipoid values may have risen in such proportion that the equilibrium was maintained and no deposits resulted.

Iodine in Therapy of Syphilis.—Burke states that Spirochaeta pallida is rich in lipoids—lecithin and globulin. The essence of the interaction between the parasite and the host is the setting free into the circulation of dead lipoprotein material. The plasma cells are the carriers of a lipoproteolytic ferment. The richer the lipoid constitution of the parasite, the greater is the affinity of the ferment for it. The lipoproteolytic ferment has an attraction toward the lipoid-rich tissues of the host. In the patient whose system is producing a satisfactory amount of lipoproteolytic ferment, the affinity between the remedial agents and the lipoid body tissues becomes reduced, because there has become interposed between the two a barrier consisting of the ferment. There is, thus, more of the arsphenamine or bismuth available to attack the parasites, which have been repelled to the tissues not so well protected. The Wassermann and other serologic tests are simply means of detecting the presence and the amount of lipoproteolytic ferment in the serum. Iodine possesses the property of being able to link itself with or to saturate the lipoids that are present in the blood of a syphilitic person. When these lipoids are saturated, they are no longer able to combine with the natural ferments and so neutralize them. From the beginning of the syphilitic infection there is a tendency for the essential lymphocyte and plasma cell reaction to progress in the direction of fibrosis. It is prudent, then, to neutralize from the earliest moment the power of the lipoids by saturating them with iodine. As a prophylactic measure, iodine should be given in every case of acute syphilis concurrently with arsphenamine or a bismuth compound, for it has no spirocheticidal effect of its own. Iodine acts not directly but indirectly on abnormal fibrous deposits, by reactivating or liberating the normal autolytic ferments of the blood. If these ferments are active there can be no excessive fibrosis, and hence the iodides are inert. They can exert their power only when the fibrosis is excessive, and for that reason no amount of iodine therapy can cause the disappearance of ordinary scar tissue, no matter what may be its origin. Such scars are reparative and not of syphilitic origin in spite of the fact that the agent that gave rise to them was Spirochaeta pallida. In the stage before a scar is formed, however, iodine and the iodides are most assuredly indispensable. No agent other than iodine possesses

the same power of rapidly causing the disappearance of gummas and gummatous infiltration. The mode of action of the spirocheticidal drugs and of iodine differs entirely, although the end results may be similar so far as fibroblastic lesions are concerned. Short of complete scarring, the denser the fibrous tissue the more need there is for the iodides to open the way for the assault by the spirocheticidal drugs. The usual manner in which iodine is administered in the treatment of syphilis is orally in the form of the potassium or the sodium salt. The author's experience has shown that the most satisfactory mode of administering iodine in the treatment of syphilis is in the colloidal form, concurrently with arsphenamine or a bismuth compound.

Treatment of Dermatophytosis with Trichophytin.—Traub and Tolmach gave 135 patients having dermatophytosis intradermal injections of trichophytin. Apparent cures were obtained in fourteen cases, but some of the patients showed early recurrence. Varying degrees of improvement were noted in fifty-eight cases. Many of these patients returned showing either no further progress or exacerbation of symptoms. In sixty-three cases no change was observed or the patient's condition was made worse by the treatment. In their experience trichophytin apparently had little if any effect on the course of dermatophytosis. In view of the publicity that this treatment has attained among general physicians, the authors believe that the comparatively small percentage of cases in which definite benefit was obtained hardly warrants such a tedious and relatively expensive course of treatment in the average case. They used two different preparations of trichophytin and did not note any difference in the results obtained. Of all their patients for whom a diagnosis of dermatophytosis had been made clinically, 21 per cent showed a negative reaction to intradermal injections of trichophytin. The value of injections of trichophytin cannot be accurately determined if effective local treatments are given concomitantly. It is well known that the use of boric acid alone can be of great help in some cases of dermatophytosis. In view of the fact that a patient with dermatophytosis frequently shows periods of remission and relapse, they feel that the reporting of cures and good results in cases of this disease should be more conservative.

Archives of Neurology and Psychiatry, Chicago

34: 481-698 (Sept.) 1935

Functional Changes in Brain of Dog After Reduction of Cerebral Blood Supply: 1. Cerebral Circulation and Development of Anastomosis After Ligation of Arteries. L. A. Andreyev, Moscow, U. S. S. R. —p. 481.
Peculiar Cells of Pick's Disease: Their Pathogenesis and Distribution in Disease. H. W. Williams, Schenectady, N. Y.—p. 508.
Hemiplegia with Leg Flexion. O. R. Langworthy, E. Highberger and Ruth Foster, Baltimore.—p. 520.
Cerebral Circulation: XLI. Vascular Responses to (A) Hypertonic Solutions and (B) Withdrawal of Cerebrospinal Fluid. H. S. Forbes and Gladys I. Nason, Boston.—p. 533.
Chordoblastoma of Basilar Plate of Skull and Ecchordosis Physaliphora Spheno-Occipitalis: Suggestions for Diagnosis and Surgical Treatment. W. P. Van Wagenen, Rochester, N. Y.—p. 548.
Cerebrospinal Hydrodynamics: VI. Correlations of Pressure of Cerebrospinal Fluid with Age, Blood Pressure and Pressure Index. J. H. Masserman, Chicago.—p. 564.
Tumors in Region of Pineal Body: Clinicopathologic Report of Three Cases. H. Zeitlin, Chicago.—p. 567.
Psychogenic Loss of Personal Identity: Amnesia. M. Abeles and P. Schilder, New York.—p. 587.
Tumors of Frontal Lobe: Anatomic and Pathologic Study. H. C. Voris, J. W. Kernohan and A. W. Adson, Rochester, Minn.—p. 605.
Effect of Endemic Malaria on Incidence of Neurosyphilis. R. J. Needles, Detroit.—p. 618.

Arch. of Physical Therapy, X-Ray, Radium, Chicago

16: 449-512 (Aug.) 1935

The Condenser Field: Improved Method of Application. F. Nagelschmidt, London, England.—p. 457.
Histamine Iontophoresis in Rheumatic and Peripheral Circulatory Disturbances. D. H. Kling, Los Angeles.—p. 466.
Newer Technic of Colon Therapy. Cora Smith King, Hollywood, Calif. —p. 474.
Short Radio Waves and Fever Therapy. W. R. Whitney and A. B. Page, Schenectady, N. Y.—p. 477.
Evaluation of Hyperpyrexia: Methods and Treatment. D. Kobak, Chicago.—p. 481.
Development of Fever Therapy in Santa Barbara Cottage Hospital. R. F. Atsatt and Luella E. Patterson, Santa Barbara, Calif.—p. 488.

Archives of Surgery, Chicago

31: 345-506 (Sept.) 1935

*Slipping of Capital Epiphysis of Femur in Adolescence. C. J. Sutro, New York.—p. 345.
Experimental Shock: Effects of Extracts from Traumatized Limbs on Blood Pressure. N. W. Roome and H. Wilson, Chicago.—p. 361.
Bactericidal Effect of Hirudin and Heparin: II. Growth of Organisms in Blood Rendered Incoagulable with Hirudin and Heparin. H. R. Mahorner and A. Ochsner, New Orleans.—p. 371.
Purulent Pericarditis: Report of Five Cases in Which Treatment Was by Pericardiotomy, and Review of Literature from April 30, 1927, to Jan. 1, 1934. A. M. Shipley and N. Winslow, Baltimore.—p. 375.
Effect of Carbon Arc Radiation on Healing of Bone. H. M. Sweeney and H. Laurens, New Orleans.—p. 395.
Clinical Aspects of Struma Lymphomatosa (Hashimoto). H. M. Clute, E. B. Eckerson and S. Warren, Boston.—p. 419.
Chronic Thyroiditis. R. K. Gilchrist, Chicago.—p. 429.
*Pharyngogenic Hematogenous Streptococcic Peritonitis. J. Felsen and A. G. Osofsky, New York.—p. 437.
Carcinoma of the Cervix Uteri: Clinical Study of Nine Hundred and Forty Cases. E. Henriksen, Baltimore.—p. 461.
Review of Urologic Surgery. A. J. Scholl, Los Angeles; E. S. Judd, Rochester, Minn.; J. Verbrugge, Antwerp, Belgium; A. B. Hepler, Seattle; R. Gutierrez, New York, and V. J. O'Conor, Chicago.—p. 477.

Slipping of Capital Epiphysis of Femur.—Sutro examined microscopically the tissues removed surgically from three patients with slipping of the capital epiphysis of the femur. They exhibited evidence that the condition is one of a fracture through the upper femoral epiphyseal plate and contiguous trabeculae of bone. No definite evidence of primary degeneration of the epiphyseal plates was seen. Variations in the weight-bearing forces at the hip joint may be a predisposing factor in the causation of the fracture. The normal tilting of the capital epiphysis, which is the result of normal developmental and mechanical forces, is the basis for the condition. Slipping of the capital epiphysis of the femur must be sharply differentiated from secondary coxa vara due to fractures, tumors, cysts or pathologic infiltrations, such as is seen in cases of Gaucher's disease. The congenital type of coxa vara may, if uncorrected in childhood, be mistaken for slipping of the capital epiphysis. In the former condition the neck is much more involved than the capital epiphysis; in the latter, changes in the angle of the neck to the femur are secondary to the position of the capital epiphysis. It is especially important to differentiate Perthes' disease from slipping of the capital epiphysis of the femur. Perthes' disease is characterized by aseptic necrosis of the capital epiphysis without any great disturbance to the epiphyseal plate. Changes in the shape of the slipped capital epiphysis are secondary to mechanical factors and not part of the disease.

Streptococcic Peritonitis.—In eight cases of streptococcic peritonitis that Felsen and Osofsky studied there was a definite antecedent history of sore throat. The average period that elapsed between the angina and the onset of acute abdominal symptoms was 5.8 days. The average duration of the disease from the time abdominal symptoms were noted until death was 4.7 days. The onset was abrupt. In three cases the condition of the throat had cleared up prior to the development of peritonitis. Three adults and five children were affected. In two of the adults the organism was nonhemolytic, but the general features were the same with both the viridans (five cases) and the nonhemolytic type (three cases). Extremely high temperatures were seen in both groups (from 107 to 107.8 F.). The initial leukocyte count was high. The Schilling counts indicated active response of the marrow. The pathologic picture in each case was essentially that of streptococcic septicemia with profound toxic effects. The lesions were most evident in the lungs, liver, spleen, intestine, mesenteric nodes and kidneys. The histopathologic pictures in general were congestive or hemorrhagic, owing to a toxic effect on the walls of the smaller vessels. Many of the clinical features noted in their studies have been described by other investigators. In most of the reported cases a hemolytic streptococcus was isolated. They believe that their group is exceptional, since five cases were of the viridans and three of the nonhemolytic type. The early appearance of diarrhea was absent in almost all their cases. The mortality was 100 per cent. In the experimental work the disease was reproduced in most of their animals by the intravenous injection of eighteen-hour broth cultures. The ease with which they did this, even with some old cultures, suggests the possibility of a selective affinity of their strains for the intestine and peritoneum.

The clinical features of the disease generally known as primary streptococcic peritonitis point to the throat as the primary source of infection in most cases in which a careful history is obtainable. The mechanism of peritoneal involvement may be directly through the blood stream, but the authors are of the opinion that it occurs through the focal hemorrhagic and ulcerative lesions in the intestine. There is ample experimental evidence to support this contention.

Georgia Medical Association Journal, Atlanta

24: 279-316 (Aug.) 1935

Maternal Mortality in Georgia During the Year 1933. Report of Committee for Study of Maternal Mortality, Atlanta.—p. 279.
Responsibility of General Practitioner in Diseases of Eye. Z. W. Jackson, Atlanta.—p. 290.
Treatment of Varicose Veins and Ulcers. C. E. Rushin, Atlanta.—p. 292.
Trend of Medical Education. R. H. Oppenheimer, Atlanta.—p. 295.
Use of Digitalis in General Practice: Its Action, Indications and Limitations. A. R. Freeman, Albany.—p. 299.
Painless Removal of Warts and Moles: Report of Cases. Marie M. Kershaw, Augusta.—p. 301.

Journal of Lab. and Clinical Medicine, St. Louis

20: 1111-1218 (Aug.) 1935

Further Observations on Patients with High Blood Sugar But No Glycosuria. R. H. Major, Kansas City, Kan.—p. 1111.
Antiseptic and Bactericidal Action of Urea. J. H. Foulger and L. Foshay, Cincinnati.—p. 1113.
*Peripheral Neuritis and Abortion Following Dinitrophenol Therapy: Report of Case. E. Epstein and H. Rosenblum, San Francisco.—p. 1118.
*Resistance of Red Blood Cells to Hemolysis in Hypotonic Solutions of Sodium Chloride: Observations in Blood Disorders. Geneva A. Daland and Katharine Worthley, Boston.—p. 1122.
Correlation of Anatomic Findings with Abnormalities of Q Wave. F. Feldman and D. Kornblum, Brooklyn.—p. 1137.
Total Sulphur of Tissue in Normal and Abnormal Growth (Mouse Carcinoma). H. Brown and J. V. Klauder, Philadelphia.—p. 1143.
*Etiologic Relationship of Amidopyrine to Agranulocytosis. F. Stenn, Chicago.—p. 1150.
Immunity Phenomena in Tuberculosis. S. A. Levinson, Chicago.—p. 1153.
Effect of High Protein Diets on Kidney Function in Dogs. L. K. Campbell, Chicago.—p. 1162.
Blastomycosis in Infant Six Months Old. J. M. Rosenthal, Allentown, Pa.—p. 1164.
Effect of Formaldehyde on Growth of Tubercle Bacilli. R. D. Herrold, Chicago.—p. 1165.
Concomitant Complete Heart Block and Bundle-Branch Block: Theoretical Discussion. E. F. Horine and M. M. Weiss, Louisville, Ky.—p. 1167.
High Blood Urea Nitrogen Not Due to Chronic Nephritis. M. G. Wohl and R. W. Brust, Philadelphia.—p. 1170.
Cardiovascular System in Protected and Unprotected Animals with Acute Diffuse Peritonitis. B. Steinberg and J. L. Kobacker, Toledo, Ohio.—p. 1180.
Observations on Intensifying Metachromatic Properties of Cresylecht Violet. B. G. R. Williams, Paris, Ill.—p. 1185.
Technic for Isolation and Preparation of Bacteriophage. Gladys Morton and Marie Wasseen, New York.—p. 1188.
Preventing Air Holes in Serum Culture Mediums. M. Edeltrude Pete, St. Paul.—p. 1193.
Bagg Albino Mice for Testing Hormones and Other Expensive Substances. J. F. McClendon and H. Street, Minneapolis.—p. 1194.
Convenient Apparatus for Rapid and Accurate Tubing of Culture Mediums. Elizabeth S. Porter and R. McBurney, University, Ala.—p. 1196.
Laboratory Study of Amidopyrine, Barbital, Phenylhydrazine and Benzene in Relation to Agranulocytic Angina. V. L. Bolton, Kansas City, Kan.—p. 1199.
*Selenium as a Catalyst in Determination of Nonprotein Nitrogen in Blood. F. Reis and H. H. Powers, Boston.—p. 1204.

Peripheral Neuritis and Abortion Following Dinitrophenol Therapy.—Epstein and Rosenblum present an instance of peripheral neuritis and abortion occurring in an obese woman following the administration of dinitrophenol. On entry to the hospital the blood gave a positive test for alpha-dinitrophenol according to the method of Bolliger. The evidence presented strongly suggests that the peripheral neuritis in this patient was due to dinitrophenol. Numerous instances of patients suffering from pains and paresthesias in the extremities while on dinitrophenol medication have been reported. The neuritis developed in this patient while the drug was being taken and disappeared within three weeks after its administration was discontinued. Compounds chemically closely related to dinitrophenol have caused true peripheral neuritis. The abortion might have been due to the drug passing through the placenta and causing the death of the fetus, or the mechanism might be analogous to the early premature separation of the placenta sometimes seen in hyperthyroidism. The absence of any discoverable etiologic factor except dinitrophenol makes it probable that the drug was responsible for the abortion. The importance of these two complications makes it advisable that the possibility of dinitrophenol causing peripheral neuritis and miscarriage be brought before the medical profession.

Resistance of Red Blood Cells to Hemolysis in Sodium Chloride Solutions.—Daland and Worthley submit a method for determining the resistance of the red blood cells to hypotonic sodium chloride. The amount of solution and the amount of red cell suspension are kept uniform throughout. The careful reading of the end-points makes it possible to observe slight variations in the resistance of the red cells. Normal maximal resistance occurs in pernicious anemia and lymphatic leukemia. Increased maximal resistance may be found in various sorts of hypochromic anemia, erythroblastic anemia (Cooley's), polycythemia rubra vera and myelogenous leukemia, depending on the degree of anemia present. In idiopathic thrombocytopenic purpura and hemophilia the changes are apparently consistent with the degree of hypochromic anemia. After splenectomy in hemolytic jaundice and occasionally in pernicious anemia during treatment, a temporary hypochromic blood picture may be accompanied by increased maximal resistance. Decreased maximal resistance is usually found in hemolytic jaundice and in advanced aplastic anemia. Normal minimal resistance may be found in moderate degrees of hypochromic anemia, including that of purpura, hemophilia, Banti's disease, sometimes in pernicious anemia and in polycythemia vera. Rarely it is normal in hemolytic jaundice. Increased minimal resistance occurs in pernicious anemia in severe relapse and in aplastic anemia. Decreased minimal resistance is usually found in hemolytic jaundice and may be found in hypochromic anemia, erythroblastic anemia, polycythemia vera and myelogenous leukemia. In general, with the exception of hemolytic jaundice and pernicious anemia in remission, the standard deviation of the cell diameters increases as the span of resistance increases. This variation in cell diameter is somewhat representative of the heterogeneity of the cell population and may be better correlated with variations in resistance than mean values. In hypochromic anemia, as the standard deviation cell diameters from the mean decrease as a result of therapy there is a progressive decrease in the span of resistance. The increased maximal resistance in hypochromic anemia, Cooley's anemia and polycythemia rubra vera is associated with a lowered mean corpuscular volume and mean corpuscular hemoglobin concentration. The latter may be important in causing flattening of the erythrocytes observed by Haden in this condition. According to the theory of Gänsslen the numerous poikilocytes should be an additional factor making for increased resistance, since they present striking deviations from the spherical form associated with a decreased resistance in hemolytic jaundice.

Relationship of Amidopyrine to Agranulocytosis.—To investigate the effect of amidopyrine on the white and differential blood count, Stenn undertook a study of 120 animals —guinea-pigs, rabbits and monkeys—over a period of eight months. Amidopyrine was given orally to one group in doses from a fraction of a grain to 3 drachms (12 Gm.), and blood and differential counts were taken daily. The symptoms were stupor, diarrhea and loss of weight. Many of the animals died of pneumonia and some from amidopyrine poisoning, but at no time was agranulocytosis observed. This work was repeated with subcutaneous, intraperitoneal and intravenous injections, but no change in the blood picture occurred. With another group an attempt was made to sensitize some of the animals treated, and these were given a rest period of one month, after which the medication was again given, still without effect. Incidentally, a pregnant guinea-pig was injected with huge doses of the drug for one and a half months, but the newborn guinea-pig and the mother both had normal blood pictures. A group of animals was bled daily until the red blood count fell between 1,500,000 and 2,000,000 and the red cells in the smear showed anisocytosis, poikilocytosis and polychromasia. Despite this preparation the white blood count failed to fall on prolonged administration of amidopyrine. In another group, small quantities of benzene in olive oil were injected subcuta-

neously until a severe neutropenia and anemia were produced. In most of the animals the white blood count fell to below 1,500. To half of this group amidopyrine was given orally, and these animals showed a more rapid rise in the white blood count and polymorphonuclear differential picture than the other animals not given the drug. A third group of animals was starved for five days and the drug administered, but without avail. A fourth group of animals was injected subcutaneously with broth cultures of Bacillus subtilis, Streptococcus viridans and Salmonella suipestifer, and one month later, with the infection still persisting, they were given heavy doses of amidopyrine orally during three weeks. The white blood counts were elevated during the period of amidopyrine treatment, and the differential counts showed a predominance of polymorphonuclear leukocytes.

Selenium in Determination of Nonprotein Nitrogen.—Reis and Powers used selenium in the determinations of nonprotein nitrogen in blood filtrates, their aim being to develop a method of digestion as rapid and as convenient as the Folin-Wu method, but without the troublesome formation of a precipitate of silica due to the action of the phosphoric acid of the digestion mixture on glass. As a result of several preliminary experiments they found that 45 per cent sulphuric acid containing a small amount of selenium could be substituted for the Folin-Wu digestion mixture with but little sacrifice of the length of digestion time and with no formation of silica precipitates. Either selenium or selenious acid may be used. The best results were obtained with a solution of 70 mg. of selenium in 200 cc. of a 45 per cent solution of sulphuric acid. The digestion is carried out in much the same way as with the Folin-Wu method. In place of the charring seen in the latter method, a rust-colored precipitate of reduced selenium appears. This dissolves as the heating is continued and the digest becomes practically colorless within thirty seconds to three minutes after the appearance of the fumes of sulphur dioxide. Five minutes of heating from the first appearance of the acid fumes is sufficient for the complete digestion of most samples of urine and for all blood filtrates, unless these filtrates, through improper preparation, contain traces of protein. In the selenium sulphuric acid method this trace of protein goes into solution but is quite difficult to digest. Usually an additional five minutes of heating completes the digestion and, if a slight brown color still persists after ten minutes of digestion, it may be disregarded as it apparently does not affect the accuracy of the determination. Analyses were done on urine and on known solutions of urea and uric acid. For the urine a value of 10.7 Gm. of nitrogen per liter was obtained as compared with 10.64 Gm. with the Folin-Wu method. The urea solution gave a value of 3.35 mg. of nitrogen per hundred cubic centimeters as compared with the calculated value of 3.5 mg. A lithium carbonate solution of uric acid on one analysis gave a value of 3.29 mg. of nitrogen per hundred cubic centimeters and, on a second analysis, 3.33 mg. as compared with a calculated value of 3.33 mg. By the use of gum ghatti the authors succeeded in nesslerizing solutions that were milky with precipitated tungstic acid, although in some cases the solution would have to stand for several minutes before all the tungstic acid was dissolved by the alkali of Nessler's solution.

Journal of Nervous and Mental Disease, New York

82: 249-372 (Sept.) 1935

Pyknolepsy. J. L. Abramson, Brooklyn.—p. 249.
Syndrome of Superior Cerebellar Artery. S. A. Sandler, Jersey City, N. J.—p. 263.
Preliminary Report on Personality Studies in Thirty Migraine Patients. Olga Knopf, New York.—p. 270.
Pathologic Changes in Tuber Cinereum in Group of Psychoses. L. O. Morgan, Cincinnati, and H. S. Gregory, Binghamton, N. Y.—p. 286.
Psychopharmacology of Sodium Amytal in Catatonia. M. W. Thorner, Norristown, Pa.—p. 299.

Michigan State M. Society Journal, Grand Rapids

34: 463-520 (Aug.) 1935

Individualized Anesthesia. R. Maurits, Grand Rapids.—p. 463.
Physiologic Postural Deformities in Contrast to Pathologic Postural Deformities. C. E. Badgley, Ann Arbor.—p. 469.
Important Integrations of the Medical School. J. E. Davis, Detroit.—p. 472.
Hematuria. W. J. Butler, Grand Rapids.—p. 479.
Puerperal Sepsis. L. A. Chrouch, Detroit.—p. 484.
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Physician's Philosophy. J. A. MacMillan, Detroit.—p. 497.
Normal Nutrition in Children. H. C. Metzger, Detroit.—p. 502.
The Doctor and Malpractice. W. J. Stapleton Jr., Detroit.—p. 504.

Military Surgeon, Washington, D. C.

77: 57-112 (Aug.) 1935

Recent Trends in Diabetes Mortality. L. I. Dublin.—p. 57.
Diabetic Coma. J. R. Darnall.—p. 75.
Standardization of Differential Leukocyte Count. A. P. Hitchens.—p. 84.
Unilateral Fused Kidney and Ectopic Right Kidney. H. C. Chenault.—p. 89.
The United States Hospital Ship Red Rover (1862-1865). L. H. Roddis.—p. 91.

Missouri State Medical Assn. Journal, St. Louis

32: 313-350 (Aug.) 1935

Advances in Renal Surgery, with Particular Reference to Nephropexy. O. S. Lowsley, New York.—p. 313.
Tuberculosis of the Genito-Urinary System. C. E. Burford, St. Louis.—p. 316.
Complications Developing After Operation for Rectal Fistula. W. R. Rainey, St. Louis.—p. 320.
Gastro-Intestinal Series, Intravenous and Oral Cholecystography: Methods in Use at the Edward Mallinckrodt Institute of Radiology. W. G. Scott, St. Louis.—p. 324.
The Present Day Rôle of Physical Therapy in Medicine. A. J. Kotkis, St. Louis.—p. 329.
Rubber Catheter Retained in Abdominal Cavity for Twenty-Six Years: Report of Case. R. Hill, St. Louis.—p. 333.

New England Journal of Medicine, Boston

213: 339-384 (Aug. 22) 1935

History of Dermatology in Boston: Chairman's Address. C. J. White, Boston.—p. 339.
*Oral Manifestations of Bismuth. F. P. McCarthy and S. O. Dexter Jr., Boston.—p. 345.
*Treatment of Psoriasis with Organic Sulphur Compound. F. M. Thurmon, Boston.—p. 353.
Diagnosis of Industrial and Nonindustrial Skin Diseases. J. G. Downing, Boston.—p. 358.
Fungus Diseases of Skin. A. M. Greenwood, Boston.—p. 363.
Report of Fatal Case of Fluoride Poisoning. L. Maletz, Hathorne, Mass.—p. 370.

Oral Manifestations of Bismuth.—McCarthy and Dexter state that the high incidence (80 per cent) of bismuth pigmentation in the mouth, in a series of 511 cases of syphilis under treatment in a hospital clinic, indicates that local oral hygiene and tartar deposits seem to represent the important factor. The contact buccal mucosal pigmentation is shown to be definitely due to irritation from the opposing gingival irritant, which is essentially tartar. Severe cases of gingival pigmentation and inflammatory reaction are relatively few, and over a period of about ten years since the preparation of insoluble bismuth salicylate in oil has been in use the number of cases of necrosis and gangrene has been extremely rare. Therefore this drug may be considered a safe therapeutic agent in the treatment of syphilis, as the presence of pigmentation and mild gingivitis seems to have no deleterious effect on the general health of the patient. A series of urinary examinations in a selected group of severe cases of pigmentation with gingivitis was negative for evidence of nephritis. Chronic nephritis was present in the two cases reported, and this condition is definitely a factor in the production of local gangrenous processes, although susceptibility to the drug undoubtedly may play an important part. Examination of the blood of a small group of patients showed no dyscrasia related to bismuth therapy. In contrast to the hospital cases studied in this series, a small control group of office patients under bismuth salicylate therapy showed a low incidence of pigmentation, about 5 per cent. Poor oral hygiene is the important factor that predisposes to pigmentation in the oral cavity. Removal of tartar deposits with improved dental hygiene hastens the disappearance of pigmentation and tends to prevent further deposits.

Treatment of Psoriasis with Organic Sulphur Compound.—Thurmon describes a solution in which the total sulphur averages 0.185 per cent by weight and is distributed possibly as 30 per cent allyl sulphide, 25 per cent polysulphide, 35 per cent allyl mercaptan, 7 per cent polymer (insoluble material) and possibly 3 per cent thio acid. Each cubic centimeter of the final solution contains about 2 mg. of sulphur. The solution may be administered with safety intravenously, intramuscularly, intracutaneously, subcutaneously, orally, by inhalation in the form of a spray and by inunction as an ointment. Blood counts and examinations of the urine, repeatedly performed on patients during and following the sulphur intravenously, have failed to reveal any abnormal change, except that occasionally patients will show a slight increase in the blood

cell count and hemoglobin, the latter increasing from five to fifteen points, the white cells to a count ranging between 9,000 and 10,000 and the red cells at times increasing as much as 1.2 million per cubic millimeter when the count is below normal. In the fifty-eight patients treated, other complications were pregnancy, diabetes mellitus, alimentary glycosuria, biliary cirrhosis, infectious jaundice, postarsphenamine hepatitis, essential hypertension, cardiorenal disease, aortic dilatation and insufficiency, generalized arteriosclerosis, and orthostatic albuminuria. The drug was administered to these and to two additional patients during the febrile period of erythema multiforme without untoward reaction. Repeated examination of the optic fundi has failed to reveal any toxic manifestation. In all cases syphilis has been ruled out as an etiologic factor. It is advisable to start each patient on treatment three times a week until ten or twelve injections of organic sulphur have been administered. For an adult, 10 cc. is the initial dose, the subsequent dosage ranging from 15 to 25 cc., depending on the response of the individual lesions. From the beginning a bland ointment is used that keeps the psoriatic scales soft and flexible, prevents and alleviates dryness, pruritus and the irritation occasionally encountered in the thickened, roughened and fissured plaques. The first change noted after the initial two or three injections is that the psoriatic areas are transformed into a vivid pink to cherry red. The process of involution continues at a pace varying with the individual response of each patient, until the areas are devoid of scales but are present as smooth, soft, pink, thin, flexible papular elevations. Finally the papules become erythematous macular areas, easily blanched with pressure, and eventually disappear when involution is completed. No pigmentation or roughness of the skin remains to mark the site of previous involvement. Intravenous organic sulphur is not a cure for psoriasis. With proper administration of the drug it is possible to control psoriasis and to effect a complete involution of the eruption. Remissions have been attained that have lasted six months. Other patients in whom complete involution of psoriasis has been accomplished have had recurrences in less than six months, but with additional treatment at the beginning of such a recurrence it is possible to render it completely under control.

New Jersey Medical Society Journal, Trenton

32: 449-506 (Aug.) 1935

Indications for Operative Intervention in Diseases of Stomach and Duodenum. G. Blackburne, Newark.—p. 455.
Physical Factors in Mental Disease. J. A. Toren, Marlboro.—p. 458.
Prophylaxis of Mental Disease. G. L. Jones, Marlboro.—p. 460.
Practical Suggestions for Management of Certain Ear, Nose and Throat Conditions. J. R. Page, New York.—p. 463.
Intestinal Obstruction. A. M. Wright, New York.—p. 465.
Treatment of Ringworm of the Feet. C. C. Carpenter, Summit.—p. 467.
Moses Maimonides the Physician. E. J. Ill, Newark.—p. 469.
Maimonides in the World of Philosophy and Metaphysics. L. S. Lang, Newark.—p. 472.
Traumatic Surgery of the Chest. S. Z. Hawkes, Newark.—p. 473.
Some New Developments in Thyroid and Parathyroid Diseases. F. H. Lahey, Boston.—p. 479.
Colitis from the Practitioner's Standpoint. A. Bassler, New York.—p. 482.
Skin Grafting. L. A. Peer, Newark.—p. 484.
Sclerosing Osteitis of Orbit: Case of Sclerosing Osteitis Following Perforation of Eyeball by Foreign Body. B. B. Adelman, Newark.—p. 487.
Care of Teeth in Pregnancy. A. W. Bingham, East Orange.—p. 488.
Infection, Susceptibility and Vaccination in Acute Anterior Poliomyelitis. J. A. Kolmer, Philadelphia.—p. 491.
Lymphogranuloma Inguinale. H. S. Talbot, East Orange.—p. 493.

New Orleans Medical and Surgical Journal

88: 139-202 (Sept.) 1935

Tribute to Dr. Joseph Goldberger. W. A. Dearman, Gulfport, Miss.—p. 139.
Classification and Treatment of Nevi. J. K. Howles, New Orleans.—p. 142.
Chronic Epidemic Encephalitis: Report of Eleven Cases with Chronic Alcoholism as Outstanding Symptom. R. C. Young, Covington, La.—p. 150.
Asthenopia and Headache, Not of Ocular Origin: Differential Diagnosis. W. T. Davis, Washington, D. C.—p. 158.
Prostate and Seminal Vesicles as Focus. E. B. Vickery, New Orleans.—p. 168.
Report of Pasteur Institute of Charity Hospital of New Orleans for Year 1934. R. D'Aunoy and J. H. Connell, New Orleans.—p. 169.
The Perforated Appendix. F. C. Shute Jr., New Orleans.—p. 171.

New York State Journal of Medicine, New York

35: 799-850 (Aug. 15) 1935

Application of Diagnostic Criteria to Treatment of Anemias. F. H. Bethell, Ann Arbor, Mich.—p. 799.
Fracture of (Navicular) Carpal Scaphoid. E. K. Cravener, Schenectady.—p. 807.
Wood Splinter in Right Orbital Cavity: Case Report. J. L. Sengstack, Huntington.—p. 809.
Poliomyelitis: Symptomatology and Treatment of Acute Poliomyelitis. Josephine B. Neal, New York.—p. 811.
Id.: Active Immunization Against Poliomyelitis. M. Brodie and W. H. Park, New York.—p. 815.
Id.: Prevention of Poliomyelitis. W. H. Park, New York.—p. 818.
Whooping Cough Vaccine as an Immunizing Agent. L. Sauer, Evanston, Ill.—p. 821.
Ultraviolet Irradiation for Secondary Anemia: Preliminary Report. R. Kovacs and I. M. Leavy, New York.—p. 825.

Philippine Islands Med. Association Journal, Manila

15: 349-406 (July) 1935

The Leprosy Problem in Philippines and Leprosy in Norway. J. O. Nolasco, Culion.—p. 349.
Intestinal Heterophydiasis with Cardiac Involvement: Contribution to Etiology of Heart Failure. C. M. Africa, Washington, D. C.; E. Y. Garcia, Binan, and W. de Leon, Manila.—p. 358.
Infant Mortality in Cebu Province from 1916 to 1932 Compared with That in Entire Philippine Islands from 1914 to 1932. C. Camomot, Cebu, Cebu.—p. 362.
Difficulties Encountered in Medical Education. K. L. Hobbs, Manila.—p. 371.
Sex Susceptibility in Leprosy. J. G. Tolentino, Mandawe, Cebu.—p. 374.
Malaria. P. Villaseñor, Lucena, Tayabas.—p. 378.

Science, New York

82: 181-202 (Aug. 30) 1935

Bodily Expression of Human Growth and Welfare. T. W. Todd, Cleveland.—p. 181.
Further Observations on Potential Rhythms of Cerebral Cortex During Sleep. A. L. Loomis, E. N. Harvey and G. Hobart, Tuxedo Park, N. Y.—p. 198.
*Intranasal or Gastro-Intestinal Portal of Entry in Poliomyelitis. J. A. Toomey, Cleveland.—p. 200.
Hemorrhagic Necrosis and Regression of Sarcoma 180. G. Shwartzman, New York.—p. 201.

Portal of Entry in Poliomyelitis.—Toomey feels that poliomyelitis in the monkey results when monkey cord virus and the toxic material in the animal's intestine combine and the combination material destroys motor cells. In the human being the causative agent usually enters the digestive system "ready made"; i. e., it is already combined and capable of producing cell destruction immediately. All the vagaries of the disease both in the Macacus rhesus monkey and in the human being, even the reason why the lumbar area usually seems to be involved first, are easily understood in the light of this theory. Much experimental and clinical work has been done that points to a relationship between the gastro-intestinal tract, the sympathetic nervous system and the production of the experimental disease. But ignoring this evidence, when virus is given either intracerebrally or intranasally, the experimental animal usually gets quadriplegia and dies. Poliomyelitis is thus produced, but even though this is poliomyelitis it is not the kind that is seen in man. The objective should be to produce the disease as it appears clinically. If poliomyelitis can be produced in the experimental animal in the same way as it is seen in the human being, it is plausible to suppose that the route taken to produce it in the monkey might be the same as that taken by the virus in the human being. The fact that intranasal inoculation is unsuccessful if the connection between the gray-fibered olfactory nerve and the central nervous system is severed is interesting. However, transection of the olfactory nerve would be of importance only if such section made it impossible to produce the disease by the gastro-intestinal tract. The author states that, if his theory as to the production of paralysis in the monkey is right, it is obvious that passive immune serum of great value cannot be obtained by injecting monkeys with the virus alone. A high-titered antiserum was rapidly produced by injecting a sheep with these combined factors—virus and enteric toxin—and recently the author has produced paralysis in a horse by injecting these combined elements.

FOREIGN

An asterisk (*) before a title indicates that the article is abstracted below. Single case reports and trials of new drugs are usually omitted.

British Medical Journal, London

2: 287-326 (Aug. 17) 1935

Pneumoconiosis, with Especial Reference to Silico-Anthracosis of Coal Miners. S. L. Cummins.—p. 287.

*Persistence of Tetanus Antitoxin in Man Following Active Immunization. P. A. T. Sneath and E. J. Kerslake.—p. 290.

Use of Poulton's Oxygen Tent in Bronchopneumonia Complicating Whooping Cough. A. Joe and J. S. Westwater.—p. 292.

Comments on Case of "Periventricular Epilepsy." T. M. Davie.—p. 293.

Rat Bite Fever. E. G. Elwell.—p. 297.

Intestinal Obstruction by Roundworms Following Administration of an Anthelmintic. J. B. Kirk and A. Y. Cantin.—p. 298.

Persistence of Tetanus Antitoxin in Man.—During the last two years, Sneath and Kerslake have determined the antitoxin content in the blood serum of a group of adults at various intervals after subcutaneous injections of tetanus toxoid. Serum was obtained from thirteen persons about two years after the last dose of the primary series of injections of tetanus toxoid. Of twelve persons in whom comparison was possible, the residual antitoxin showed no evidence of having fallen below that shown from twelve to fifteen months after the last dose. Three of the group retained at least 0.1 unit of tetanus antitoxin per cubic centimeter. In view of the limitation to accurate interpretation of the tests, the apparent increases in antitoxin titer shown by three of the group during the second year cannot be accepted without reserve. If subclinical infection by Clostridium tetani were a possible explanation of such a delayed antitoxin increase, acting in this instance as a secondary stimulus, it is probable that a higher residual titer would be found than was shown. It is therefore considered that the apparent increase in antitoxin titer over this period is probably not significant. A fourth dose of toxoid was given to these twelve persons and serum was obtained at intervals of a week and a month thereafter. Within a week an increase in antitoxin titer occurred in the order of from ten to five hundred times more than the residual level shown two years after the primary series. After a month, five showed a further increase in antitoxin titer, four showed no appreciable change, two showed a definite fall and one serum was not tested. Eleven persons developed tetanus antitoxin in amounts of between 0.1 and 10 units per cubic centimeter. One subject, whose titer did not rise above 0.025 unit, has shown a poor antitoxin response throughout. Nevertheless, the increase is similar in trend to that of the other members of the group. A fourth subcutaneous dose of 1 cc. of tetanus toxoid given to fourteen persons about a year after a primary series of three doses has been shown to occasion a rapid increase of antitoxin within a week, thirteen of the group attaining or exceeding an antitoxin titer of from 0.1 to 0.25 unit per cubic centimeter, which level was maintained after a month. A year later one subject had retained approximately the same antitoxin titer as was found a month after the fourth dose, whereas in the others the titer had definitely fallen. Nine persons retained antitoxin in amounts varying from 0.1 to 0.75 unit per cubic centimeter, these residual levels in general being substantially in excess of those found a year after the initial doses.

Journal of Laryngology and Otology, London

50: 569-648 (Aug.) 1935

*Anatomic Investigation of Blood Vessels of Lateral Nasal Wall and Their Relation to Turbinates and Sinuses. H. H. Burnham.—p. 569.

Treatment of Acute Suppurative Otitis Media by Syringing with Alcohol. V. Schmidt.—p. 594.

Relation of Blood Vessels of Nasal Wall to Sinuses.—Burnham investigated the course and relations of the blood vessels of the lateral nasal wall and accessory sinuses. Comparatively large bony canals are present in the central part of the middle and posterior half of the inferior turbinate bones. They contain terminal branches of the turbinate arteries, veins (jacket plexus) which form an essential part of the "venous pathways," and nerves. The veins and nerves are enclosed in the periosteum that surrounds the artery. Owing to the delicate structure of the canals, they may be almost entirely destroyed in a macerated specimen and this may account for the vagueness of the descriptions. Three definite canals are present in the posterior half of the inferior turbinate. The uppermost contains vessels for the antrum, and its connection with the latter through the "uncinate aperture" is described. This aperture is a constant naso-antral bony opening, inferior to the uncinate process, through which the antrum obtains a large part of its blood supply. In the central third of the middle turbinate, a single canal terminates anteriorly in three smaller divisions. The term "venous pathways" is used to indicate the large channels of veins that carry the blood over the lateral nasal wall to the sphenopalatine foramen. They are the inferior, middle and superior turbinate venous pathways. Each pathway is made up of erectile vessels, which pass into either periosteal or "intra-osseous" veins, and each of these joins the jacket plexus of veins within the bony canals of the turbinate. They then leave the canals, passing into periosteal veins again before reaching the sphenopalatine foramen. The intra-osseous veins are mainly present in minute bony canals in the anterior half of the middle and inferior turbinates. The erectile tissue empties directly into these veins; very few arterioles are associated with them. The erectile tissue also passes into the periosteal vessels that accompany the larger arteries. These periosteal veins enter the large bony canals in the posterior half of the turbinate and are continuous with the jacket plexus which the latter contains. A few of the periosteal veins also accompany the arteries that pass over the lateral nasal wall to the sphenopalatine foramen. They are, however, of distinctly minor importance compared to the large bony canal channels. The superficial veins of the lateral nasal wall present such sharp turns and twists in their courses that they may be described as convoluted. This is particularly true of the erectile tissue areas. The periosteal veins, on the other hand, both those found within the large turbinate canals and those on the surface, have a more wavy course with much fewer and less abrupt turns. The veins of the sinuses are of the periosteal type and, from the plexus about the ostium, take a comparatively straight course into the sinus. This ostial plexus is but a few millimeters in width, except in the case of the antrum, in which it covers a large part of the medial wall. The vessels pass into the sinus and either end in capillaries or are conjoined to collateral branches. In the sinus, no definite superficial layer of veins has been found and a distinct contrast is evident between the straight coursed veins of the sinus and the convoluted ones of the lateral nasal wall. The erectile (cavernous) tissue vessels present pouches or pockets in their walls, continuous with the lumen of the vessel but enlarging it considerably.

Journal Obst. & Gynaec. of Brit. Empire, Manchester

42: 577-732 (Aug.) 1935

Upper Urinary Tract in Pregnancy and Puerperium, with Especial Reference to Pyelitis of Pregnancy. D. Baird.—p. 577.

Muscular Spasm in Fetus: Factor in Production of Malpresentation. G. F. Gibberd.—p. 596.

Significance of Shape of Fetal Head in Mechanism of Labor. E. Rydberg.—p. 600.

Survey of Results of Treatment of Prolapse and Retroversion in Royal Free Hospital from 1926 to 1933. Margaret Salmond, in conjunction with Gertrude Dearnley.—p. 623.

Vaginal Metastasis in Hypernephroma. K. Bowes.—p. 630.

Pernocton-Hyoscine Twilight Sleep: Review of Thirty Cases. A. M. Claye.—p. 636.

Polyneuritis of Pregnancy. R. K. Ford.—p. 641.

Zondek-Aschheim Pregnancy Test, with Especial Reference to Reaction I. Mabel F. Potter.—p. 646.

Four Ectopic Pregnancies in Same Woman. J. B. Dawson.—p. 651.

Perforation of Diverticulum of Pelvic Colon into Ovarian Cyst. D. J. Harries.—p. 653.

Abdominal Belt as Mechanical Aid Before and During Labor. A. Danby.—p. 655.

Hydatidiform Mole: Spontaneous Perforation of Uterus. H. I. McClure.—p. 663.

Japanese Journal of Obstetrics and Gynecology, Kyoto

18: 185-300 (June) 1935

Extracorporeal Culture of Uterine Cancer: Attempt to Improve Culture Medium. I. Narita.—p. 186.

Experimental Study of Effect of Thyroid Function on Growth of Malignant Tumors: Parts I, II, III, IV, V and VI. S. Nishida.—p. 195.

Histologic Investigation of Fetal Kidney: Part II. Experimental Study of Renal Disturbance in Fetal Period. S. Tsuda.—p. 233.

Cytologic Study of Living Body Irradiated with Hard Ray: Parts II, III and IV. T. Saito.—p. 237.

Presse Médicale, Paris

43: 1265-1280 (Aug. 10) 1935

*Relative Value of Cholecystography and Gallbladder Excretion Test in Diagnosis of Cholecystitis. M. Chiray, I. Pavel and A. Lomon. —p. 1265.
Pleural Pressure. Baillet.—p. 1267.

Cholecystography and Gallbladder Excretion Test in Diagnosis of Cholecystitis.—Chiray and his collaborators studied the relations of the Meltzer-Lyon test and cholecystography to disease of the gallbladder and biliary tract. It is obvious that a negative cholecystogram can be related to a pathologic state of three organs without counting the sphincter of Oddi, whereas the negative Meltzer-Lyon test indicates the gallbladder alone as the cause. Thus cholecystography is principally valuable when positive, while the Meltzer-Lyon test has theoretically the same value for positive and negative tests. In the biologic study of gallbladder disease by those means, three possibilities arise. When both duodenal drainage and the cholecystogram give a normal response, it is almost certain that the gallbladder is not involved. When both tests are abnormal, it is almost equally certain that there is a diseased gallbladder present. When the two tests are divergent, the diagnosis involves real difficulties. The most common difference is when cholecystography gives a negative or questionable image and the Meltzer-Lyon test shows a "B bile" of variable character. In these cases the latter indicates the true diagnosis. In the inverse case, when cholecystography gives a positive result and duodenal drainage is negative, pericholecystitis is most often the cause. In conclusion the authors state that the two tests are not always superimposable and that one cannot be substituted for the other, but they are complementary and the employment of both does much to increase the diagnostic accuracy to an extent previously unknown.

Minerva Medica, Turin

2: 305-336 (Sept. 8) 1935

Tuberculous Meningitis in Adult Patient Presenting Pulmonary Tuberculosis. L. Ferrannini.—p. 305.
Pernicious Anemia and Diabetes with Especial Attention to Deficiency of Vitamin B_9. A. Archi.—p. 309.
Experimental Research on Lymphography. E. Lucca.—p. 314.
*Influence of Adrenal Cortex Extract on Bactericidal Power of Blood. E. Caldarera.—p. 320.
Skin Reaction to Gold and Gold Therapy in Pulmonary Tuberculosis: Skin Reaction to Gold with Principal Gold Salts. G. Chiucini and A. Aradas.—p. 323.

Influence of Adrenal Cortex Extract on Bactericidal Power of Blood.—Caldarera administered two subcutaneous injections of 0.2 cc. of adrenal cortex extract twenty-four hours apart to a number of rabbits. The bactericidal power of the blood was determined in each animal before treatment and two, eight and twenty-four hours after the second injection. Blood was taken from each animal and added to an equal amount of physiologic solution of sodium chloride citrated at 1.5 per cent. One cubic centimeter of the citrated blood was placed in each of two tubes. One drop of a 1:40,000 suspension of Staphylococcus aureus was added to the blood in one tube and a similar proportion of colon bacillus to the other. The tubes were placed in an incubator at 37 C. for three hours, after which the blood plates were prepared. At the same time a control plate was prepared with a mixture of 1 cc. of physiologic solution of sodium chloride citrated at 1.5 per cent with one drop of bacterial suspension. The number of colonies developing on the control plate and on the blood plate were compared. The author found that the bactericidal power of the blood is constantly increased by the use of adrenal cortex extract. Plates of blood obtained two hours after injection of extract showed a diminution of bactericidal power (greater development of colonies), while those obtained after eight hours showed a marked increase. Those obtained after twenty-four hours showed a tendency of the bactericidal power to return to normal. This tendency varies according to the microorganism used. The bacterial index of Staphylococcus aureus, for example, was higher after twenty-four hours than in the beginning, while the bacterial index of colon bacillus after twenty-four hours was on a par with or lower than the initial values.

Prensa Médica Argentina, Buenos Aires

22: 1607-1654 (Aug. 21) 1935

*Benign Spontaneous Pneumothorax Due to Rupture of Subpleural Bubbles. M. R. Castex and E. S. Mazzei.—p. 1607.
Pericardial Friction Rub and Its Semeiologic Value in Topographic Diagnosis of Myocardial Infarct. G. Bosco.—p. 1624.
Anatomoclinical Forms of Pulmonary Syphilis in Adults. D. Vivoli. —p. 1627.
Von Jacksch's Anemia and Mikulicz's Syndrome: Case. C. Zubizarreta and J. E. Herran.—p. 1642.

Spontaneous Pneumothorax Due to Rupture of Subpleural Bubbles.—Castex and Mazzei report twelve cases of benign spontaneous pneumothorax due to rupture of subpleural bubbles. They state that the disease develops in persons (especially men) ranging in age between 22 and 30. All the authors' patients were men. There were no symptoms of tuberculosis in evolution. The disease is afebrile and lasts about four weeks, ending in recovery without leaving signs of any pathologic process that can be detected in the roentgen examination of the lung. There is slight or no pleural effusion. The diagnosis is confirmed if the bubbles are visible in the roentgenogram, as happened in three of the authors' cases. Active or latent tuberculosis does not play a part in the etiopathogenesis of the disease. The bubbles, the size and number of which are variable, may be located in different parts of the lung, such as the apex, borders, aspects and fissures. As a rule they originate in pleural scars. Inflammatory processes and congenital malformation of the lung have been noted. The treatment is one of expectation and consists chiefly in rest and symptomatic remedies. If there are complications, such as circulatory shock and acute anoxemia, the employment of analeptics and of oxygen is advised. Only in one of the authors' cases was part of the pleural air removed.

Deutsche medizinische Wochenschrift, Leipzig

61: 1385-1424 (Aug. 30) 1935. Partial Index

Diagnosis of Schizophrenia and Cyclothymia. K. Schneider.—p. 1385.
Organization of Campaign Against Cancer. A. Hintze.—p. 1390.
Protein Requirements of Human Beings. B. Süsskind.—p. 1393.
*Angioid Streaks. W. Reichling.—p. 1394.
Erroneous Diagnoses in Convulsions During Childhood. E. Hässler. —p. 1396.
*Serologic Differential Diagnosis Between Brucella Abortus and Brucella Melitensis Infection in Human Subjects. H. Habs and Lena Sievert. —p. 1398.

Angioid Streaks—Reichling observed three patients with angioid streaks in the retina. Two of these patients had a careful dermatologic examination, which, however, gave negative results. Although the presence of pseudoxanthoma elasticum could not be proved in his cases, the author is inclined to agree with other observers who consider the disorder a systemic disease of the elastic substance.

Differentional Diagnosis of Brucella Abortus and Brucella Melitensis.—Habs and Sievert point out that a differentiation of the various types of Brucella is desirable from the epidemiologic point of view. In cases in which the causal organism can be cultured, the differentiation is rather simple. However, in practice, the specific diagnosis is usually based on the outcome of the immunity reactions, particularly the agglutination. Formerly, the agglutination test did not permit a differentiation. But, since the studies carried out by Wilson and his collaborators have provided a better understanding of the antigen structure of the various brucella types, the authors considered new tests advisable. Wilson had demonstrated that only the smooth strains should be used, because the rough strains produce disturbances. He had found also that the difference between abortus and melitensis groups is not that in addition to a common group antigen they also each have a specific antigen but rather that both groups contain two identical antigens in differing quantities. Thus there is really only a quantitative difference between the two types. The authors state that, although their observations differed slightly from those of Wilson, they were able to corroborate the fact that it is possible to differentiate the melitensis and abortus types by quantitatively graded absorption experiments. Former experiments had for an object the classification of the brucella group into different types by means of rabbit immune serum. In the experiments described here, the opposite method was employed; that is, by means of known strains it was attempted to determine

by absorption experiments the agglutinin content of the serum of patients and thus find whether the patients had a Brucella abortus or a Brucella melitensis infection. At first the authors followed the technic suggested by Wilson, but later they modified it in various ways. They give several tabular reports, indicating the observations on various serums. On the first, a melitensis serum, absorption experiments were made with a melitensis strain on the one hand and with an abortus strain on the other. The absorbed serum was further combined with another abortus strain, with a suis strain and with two additional melitensis strains. The table indicates that, following the absorption with the melitensis strain, none of the abortus strains became agglutinated. Following absorption with the abortus strain, however, only the agglutinins for the abortus strains and the suis strains were withdrawn, whereas the melitensis strains were still agglutinated up to one fourth of the original serum titer. Thus it is proved that the serum was from a patient with Brucella melitensis. The authors describe tests on several other serums.

Medizinische Klinik, Berlin

31: 1125-1156 (Aug. 30) 1935. Partial Index

Myasthenia Pseudoparalytica. H. Curschmann.—p. 1125.
*Function of Thyroid and Heart. G. W. Parade.—p. 1130.
Biomotor of Eisenmenger as Life Saving Apparatus in Poliomyelitic Respiratory Paralysis. F. Hamburger.—p. 1132.
Glyceryl Trinitrate in Prophylaxis of Angina Pectoris. F. Kisch.—p. 1133.
Mistakes in Diagnosis and Treatment of Cystitis and Pyelitis. W. Pollak.—p. 1136.

Function of Thyroid and Heart.—Parade discusses the circulatory mechanism in case of dysfunction of the thyroid. He emphasizes that the thyroid is an important link in an interconnected system in which the sympathetic centers of the diencephalon, the hypophysis, the sympathetic nervous system, the adrenal cortex and other incretory organs play a part. In discussing the effects produced by the hypofunction of the thyroid on the heart and the circulation, he points out that during starvation there develops a bradycardia as manifestation of a hypofunction of the thyroid and that hibernation in animals is accompanied by an involution of the thyroid and a great reduction in the frequency of the pulse. In human myxedema one observes bradycardia and a slowing down of the circulation and of the metabolic processes. If, however, under the influence of thyroid medication the myxedema symptoms disappear, the heart action likewise improves. The close relation between thyroid and heart is proved further by the fact that the administration of thyroid extract is followed by acceleration of the pulse and other cardiac symptoms if too large doses are given. The author discusses the cardiac function in hyperthyroidism. He shows that the increased rate of the basal metabolism in exophthalmic goiter is accompanied by accelerated circulation. He points out that absolute arrhythmia with auricular fibrillation is relatively frequent in older persons with exophthalmic goiter. He emphasizes that, if old persons have a cardiac insufficiency of obscure etiology, particularly if the insufficiency does not respond to digitalis and is accompanied by auricular fibrillation, a thyrogenic origin should be considered and the proper therapeutic measures should be taken to counteract the thyrotoxicosis.

31: 1157-1188 (Sept. 6) 1935. Partial Index

Liver and Diabetes. W. Falta.—p. 1157.
Therapeutic Actions of Carbon Dioxide. R. Cobet.—p. 1160.
*Nervous Disturbances in Pernicious Anemia with Especial Consideration of Their Amenability to Therapy. G. Straube.—p. 1164.
Paroxysmal Ventricular Tachycardia in Case of Pericardial Calcification. R. Fischer and K. Wasserbrenner.—p. 1171.
*Does Positive Outcome of Agglutination Test Prove the Presence of Brucella Abortus Infection. W. Hauptmann.—p. 1174.

Nervous Disturbances in Pernicious Anemia.—Straube summarizes his observations on the nervous symptoms of patients with pernicious anemia, compares his observations on certain points with those of other investigators and pays especial attention to the therapeutic aspects. In discussing the etiology, he expresses the opinion that funicular myelitis as well as other organic nervous disturbances are not the result of the anemia, for they have been known to precede the anemia, but that the anemic as well as the spinal symptoms may have a common cause. After discussing the histologic aspects and the incidence of the spinal symptoms, he gives his attention to the question whether funicular myelitis has become more frequent since the era of liver therapy. He thinks that the patients survive longer, which allows more time for the development of funicular myelitis. In reviewing the literature on the therapeutic amenability of the spinal symptoms, the author observed great discrepancies not only in the selection of the therapeutic method but also in the estimation of the results. However, he found that all those who employed liver therapy in a large number of cases agree that funicular myelitis is not favorably influenced by the liver therapy. At the author's clinic several hundred cases were observed. The liver therapy had the desired effect on the blood status and the general symptoms, but, in spite of the fact that excessive doses of liver were given in the hope that a therapeutic effect might be exerted on the spinal symptoms, the results were negative. To be sure, in some instances the paresthesias and the disturbances of sensitivity improved, but these improvements were, as a rule, not lasting. The author concludes that the spinal symptoms which accompany pernicious anemia cannot be cured and cannot even be prevented. However, he does not think that treatment should be entirely omitted but suggests that the combined administration of strychnine, arsenic, phosphorus and liver may eventually be effective. Moreover, he thinks that general tonicizing measures, such as walking exercises, massage and hydrotherapy, might be helpful.

The Agglutination Test and Brucella Abortus Infection.—Hauptmann points out that the occurrence of positive Brucella abortus agglutination reactions in healthy persons and in patients with other disorders, which he observed in the course of tests on 2,315 serums, limits the diagnostic value of the agglutination test. The existence of a Brucella abortus infection is the more likely, the higher the agglutination titer is above the 1:100 ratio. However, there is no definite boundary line that applies to all cases. The author stresses that the positive outcome of the Brucella abortus agglutination test should not induce the diagnostician to give up the thought that typhoid or paratyphoid might be present. Complementary examinations will be necessary in order to reach a definite diagnosis.

Münchener medizinische Wochenschrift, Munich

82: 1389-1430 (Aug. 30) 1935. Partial Index

Serum Anaphylaxis After Injections of Tetanus Antitoxin. E. Mackuth.—p. 1392.
*Action of Sulphur in Experimental Carbon Monoxide Poisoning. K. Voit and H. H. Schmidt.—p. 1393.
Substitute for Anal Sphincter Produced by Thermocauterization. L. Moszkowicz.—p. 1395.
*Congo Red in Treatment of Intestinal Hemorrhage During Typhoid. F. Szirmai.—p. 1403.

Sulphur in Experimental Carbon Monoxide Poisoning.—Voit and Schmidt direct attention to studies conducted by Vita and Salmoiraghi. In animal experiments on the action of colloidal sulphur in carbon monoxide poisoning, these investigators were able to show that guinea-pigs treated with sulphur survived carbon monoxide poisoning and recovered in a relatively short time, while the control animals died. When the animals were exposed to fatal doses, those which had received prophylactic injections of sulphur survived three times as long as those which had not. In spectroscopic studies on the detoxication mechanism in experimental carbon monoxide poisoning, it had been determined that at the time the signs of poisoning disappear there appears a blood spectrum that greatly resembles the spectrum of carbon oxysulphide in the blood. From this observation it was deduced that carbon oxysulphide is formed in the blood during carbon monoxide poisoning and that this is a detoxication process. It was thought that the injection of sulphur might accelerate the formation of carbon oxysulphide. The authors decided to investigate whether the prophylactic treatment with sulphur would prevent the development of erythrocytosis, leukocytosis and hyperglycemia, which frequently develop in the course of carbon monoxide poisoning. They studied the action of sulphur on acute as well as on chronic cases. They found that the injection of sulphur prevents the development of hyperglycemia and of erythrocytosis. The observation of Vita and Salmoiraghi that the animals

treated with sulphur have a greater resistance to carbon monoxide poisoning could be corroborated in the majority of animals, but not in all.

Congo Red in Treatment of Intestinal Hemorrhage.—Szirmai considers severe intestinal hemorrhage one of the gravest complications of typhoid, and, in order to control it in several cases of typhoid that were under his treatment, he followed Pelyi's suggestion and employed intravenous injections of congo red. He points out that congo red belongs to the electronegative, neutral, high-molecular dyestuffs. In experiments it was proved to be nontoxic, and it was found to increase the blood platelets, the monocytes and the coagulability of the blood. The author employed congo red in ten typhoid patients with severe intestinal hemorrhages. In four cases a single intravenous injection of 10 cc. of a 1 per cent solution stopped the hemorrhage, while in the other cases two or three injections were necessary. The repeated injections are given at intervals of twelve or twenty-four hours. Aside from mild attacks of chills that developed in two patients, there were no harmful results from the congo red.

Wiener klinische Wochenschrift, Vienna

48: 1103-1126 (Sept. 6) 1935

Relations Between Heredity and Endocrinology. J. Bauer.—p. 1103.
Electrohygiene. S. Jellinek.—p. 1107.
*Clinical Aspects and Differential Diagnosis of Foreign Body Tumors of Colon. M. D. Manizade.—p. 1111.
Casuistic Contribution to Cerebral Surgery. J. Hohenwallner.—p. 1114.
*Influence of Diuretics on Alkali Reserves of Blood. G. Gottsegen.—p. 1116.

Foreign Body Tumors of Colon.—Manizade describes a case of foreign body tumor of the colon presenting the aspects of a malignant neoplasm. Not until after a careful anatomic examination was it possible to exclude the existence of a malignant neoplasm, and this examination revealed also the real cause of the chronic inflammatory process; namely, a small foreign body (chicken bone). Although it is generally assumed that swallowed foreign bodies are discharged from the intestine by the natural route as soon as they have passed the duodenal flexure and the ileocecal valve, a review of the literature discloses that occasionally inflammatory tumors arise on the basis of a foreign body lodged in the lower portion of the intestine. It appears that the sigmoid is most subject to such processes, since this section of the intestine favors the lodging of foreign bodies because of its flexures and the retarded passage of the intestinal contents. However, another observer has suggested that the size of the lumen plays a part in the lodging of and perforation by a foreign body. The author, on the other hand, cites a case in which retardation of the passage and flexure of the intestinal tube were responsible for perforation by a foreign body. The peculiar postoperative course in the first patient, that is, the recurrent inflammatory tumor, induced the author to search for the causes of this behavior. He points out that Weltmann's coagulation reaction is helpful not only in differentiating chronic inflammatory tumors and neoplasms but also in detecting a change in the protein coagulation, which in turn would explain the development of such hyperplastic tumors. In the reported case the Weltmann reaction disclosed an extremely wide coagulation band. This outcome of the test favors definitely a chronic productive process and militates against a neoplasm but also indicates why the foreign body gave rise to such extensive inflammatory proliferation. The author points out that persons with dental prostheses, particularly if the prostheses cover the entire palate, readily swallow comparatively large bodies without noticing them.

Influence of Diuretics on Alkali Reserve of Blood.—Gottsegen thinks that it cannot be doubted that changes in the acid-base equilibrium are of great importance for the water economy of the organism. He cites the opinions of several other investigators and then reports studies carried out by himself. He found that mercury diuretics (salyrgan and others), euphylline and sodium dehydrocholate increase the alkali reserve of the blood. The mercury compounds have the strongest action in this respect, the bile acid the weakest. The combination of mercury salts with sodium dehydrocholate changes nothing in regard to the development or the extent of the alkalosis. Ammonium chloride inhibits the alkalosis only if its administration is continued following the injection of the diuretic. If it is given only before, the alkali reserve increases in the same manner as if only the mercury compound had been given. The diuretic action of ammonium chloride cannot be ascribed entirely to the acidosis that develops; but it may be assumed that various changes in the internal milieu play a part.

Zentralblatt für Chirurgie, Leipzig

62: 2049-2112 (Aug. 31) 1935. Partial Index

Notomelus. E. König.—p. 2050.
*Results of Serum Treatment in Peritonitis. O. Kapel.—p. 2053.
*Thrombophlebitis Migrans (Saltans) and Its Etiology. J. Jáki.—p. 2056.
Interesting and Rare Complications of Adhesion Ileus. A. Adler.—p. 2059.
Cancer Caused by Arsenic: Case. S. Miyaji.—p. 2063.

Serum Treatment of Peritonitis.—Kapel says that he has employed a combination of a serum obtained by the immunization of horses with the toxins of selected colon strains with a so-called peritonitis serum prepared in the same manner by means of Bacillus perfringens. The ampules of colon serum contained 25 cc. and the ampules of perfringens serum contained 20 cc. In severe cases the author administers the first dose intravenously, together with an infusion of a sodium chloride solution, while the patient is still under the anesthetic. The same dose is also given intramuscularly. On the following five days, the patient is given daily one ampule of each of the two serums intramuscularly. In the case of children, the injections are given for only three days. In cases in which the general condition is not noticeably impaired, the intravenous injection is omitted and only the intramuscular injections are given. The author employed serotherapy in all cases of diffuse, free perforation peritonitis, in the majority of cases of localized perforation peritonitis, in cases in which there was an accumulation of pus around the vermiform process and finally in cases in which large portions of the appendix were gangrenous so that the bacteria could penetrate. In investigating the results of this serotherapy, the author finds that it produced no changes in the total mortality rate. Nevertheless, he gained the impression that the serum exerted a beneficial effect in patients who recovered, for the signs of general intoxication were less severe than is otherwise the case; the intestinal function was reestablished in a shorter time, and the recovery was more rapid. The serotherapy had the disadvantage that in some instances it resulted in arthralgias, exanthems, edemas and fever, but these disturbances were only temporary. He concludes that further experimental studies will be necessary in order to determine whether these serums really contain antibodies. He is inclined to believe that these serums are only antibacterial, not antitoxic.

Thrombophlebitis Migrans (Saltans).—According to Jáki, thrombophlebitis migrans differs from ordinary thrombophlebitis in the manner in which it appears and in its clinical symptoms. It usually appears in the superficial, peripheral veins. Another characteristic is that it appears in widely distant regions. The author points out that the term "migrans" is really not quite suitable, for the relapsing thrombophletitis generally does not spread progressively, although this too is possible, but advances by leaps, leaving healthy sections in the same vein and suddenly appearing in a distant vein. For this reason he thinks that the term thrombophlebitis "saltans" would be more fitting. As a rule, thrombophlebitis migrans (saltans) appears without preliminary symptoms, but occasionally spasm-like pains precede it. He reports two cases; in describing the histologic aspects, he points out that all layers of the venous wall show signs of inflammation. Many investigators consider the etiology unknown. He rejects Buerger's opinion, according to which thrombophlebitis migrans is supposedly an accompanying symptom of thrombo-angiitis obliterans. Other investigators consider syphilis or tuberculosis a causal factor, but they do not furnish definite proof for this. The author thinks that the clinical aspects indicate that an infection of low virulence is the cause. However, in addition to an infectious origin, a characteristic reaction of the veins plays a part. It is chiefly the latter factor that differentiates the process from the ordinary type of thrombophlebitis. He is of the opinion that thrombophlebitis migrans (saltans) is an allergic problem. He thinks that a hypersensi-

tivity is involved and that a disturbance in the sympathetic nervous system plays a part in this hypersensitivity. The infectious cause should be given especial attention. In the reported cases a latent infectious focus in the teeth was the eliciting cause. The search for and the elimination of the latent infectious focus is one of the most important factors in the treatment.

62: 2113-2160 (Sept. 7) 1935. Partial Index

*Disinfection of Operative Field by Means of Solution of Formaldehyde. E. Borchers.—p. 2114.
Innervation of Vena Saphena with Regard to Operations on Varicose Veins. Mathilde Ambrus.—p. 2120.
*Computation of Basal Metabolic Rate from Pulse Frequency and Pulse Pressure by Means of Read's Formula. Annemarie Oettinger.—p. 2124.
Case of Congenital Sarcoma. E. Kubanyi.—p. 2126.

Disinfection by Solution of Formaldehyde. — After pointing out that the use of tincture of iodine in the disinfection of the operative field has certain disadvantages, Borchers states that for about nine years he has used a 5 or 10 per cent alcoholic solution of formaldehyde. He proved the suitability of the formaldehyde preparation in practice and by experiments. He describes comparative tests, and in summarizing his observations he states that (1) the immediate influence of the alcoholic solution of formaldehyde on the bacterial content of the skin is better than that of tincture of iodine; (2) its lasting action on the bacteria of the skin is better than that of tincture of iodine; (3) injuries of the skin (dermatitis, necrosis) are almost never observed, if the application of the formaldehyde has been suitable; (4) exudation from the puncture channel occurs only in a small number of cases, and (5) solution of formaldehyde is much less expensive than tincture of iodine.

Computation of Basal Metabolic Rate.—Oettinger calls attention to the fact that Read's formula makes the determination of the basal metabolic rate comparatively simple in that it does not require the use of a complicated apparatus but computes the metabolic rate according to the following formula: Basal metabolic rate $= 0.75$ (pulse rate $+ 0.74$ pulse pressure) $- 72$. In order to determine the reliability of the formula, the author made comparative tests on fifty-nine persons; that is, she determined the basal metabolic rate by means of an apparatus and also computed the rate according to Read's formula. In the majority of cases she found only slight discrepancies. She also was able to corroborate Read's statement that his formula does not give reliable results in persons less than 15 and more than 55 years of age, in those with a blood pressure in excess of 160 mm. of mercury, in patients with severe disturbances in the cardiovascular-renal system and in severe cardiac lesions. She concludes that, even with these limitations, the formula is valuable for use in the clinic and for the general practitioner.

Zentralblatt für Gynäkologie, Leipzig

59: 2049-2096 (Aug. 31) 1935

Irritability of Uterine Muscle and Galvanic Irritability of Nerve-Muscle System. E. Klaften and Z. Ruffel.—p. 2050.
*Experimental Studies on Influence of Iron and Phosphorus on Pregnant Organism and Fetus. R. L. Schub.—p. 2058.
Hormone Therapy for Increase in Milk Secretion. K. Völz.—p. 2061.
Treatment of Umbilical Hernia in the New-Born. J. Oberholzer.—p. 2062.
Does Renewed Surgical Treatment or Delivery Involve Danger of New Thrombosis in Patients Who Have Passed Through One Thrombosis? M. Mátyás.—p. 2066.

Influence of Iron and Phosphorus on Pregnant Organism.—Schub made studies on ninety-six pregnant white rats. One half of this number of animals served as controls. The latter were given food that was deficient in iron and phosphorus. The other animals received in addition to the same food organic iron and organic phosphorus. Every third day the animals were carefully weighed. Already on the fifth day the experimental animals showed an increase over the controls. The increase in weight during the entire period of observation was 38 Gm. in the rats that received no addition of iron or phosphorus and 63 Gm. in the animals that received the addition. The offspring of the control animals weighed 4.4 Gm. and that of the experimental animals 5.18 Gm. After delivery and the cessation of the iron and phosphorus therapy, the experimental animals lost weight less rapidly than did the controls.

Acta Medica Scandinavica, Stockholm

85: 493-597 (Aug. 28) 1935. Partial Index

Aim and Prospects of Medicinal Treatment of Cancer. R. Roosen.—p. 499.
Alternating Spontaneous Pneumothorax in Apparently Healthy Persons. S. Grapengiesser.—p. 505.
"Duodenal Regurgitation" Versus "Electrolyte Diffusion" in Gastric Juice. T. Teorell.—p. 518.
Correlation Between Acidity and Total Chloride Amount in Clinical Test Meals. P. J. Nordenfelt and T. Teorell.—p. 525.
Thallium Poisoning: Case. A. Gjertz.—p. 531.
*Pulmonary Bilharziasis Simulating Tuberculosis. F. Mainzer.—p. 538.
Effect of Vitamin C Therapy on Lowered Capillary Resistance in Patients with Gastric Achylia. P. Schultzer and O. Griis.—p. 563.
Blood Proteins and Sedimentation of Erythrocytes. E. Kylin.—p. 574.

Pulmonary Bilharziasis Simulating Tuberculosis. — Mainzer reports five cases of a chronic infiltrative disorder of the lung which simulated tuberculosis but which was accompanied by an enlargement of the spleen and an excessive eosinophilia and responded to treatment with antimony. The eosinophilia, the treatment, the simultaneous presence of a urinary and intestinal bilharziasis in one case (and a similar report in the literature) and the clinical and experimental aspects of a pulmonary infection in the course of the development of different forms of schistosomiasis favor the assumption that the disorder was a pulmonary schistosomiasis. The author differentiates the described cases of bilharziasis into three groups. The first group, represented by one case, can be designated as bronchial asthma produced by Bilharzia. The second and third groups are pulmonary bilharziasis in the strict sense of the term. The second group includes the acute cases of pulmonary bilharziasis. In this form the foci may completely disappear after the treatment. The third group includes the chronic cases of pulmonary bilharziasis in which a pulmonary cirrhosis remains after the treatment. The author thinks that two reports in the literature under the headings of "familial eosinophilia" and "persistent eosinophilia with enlargement of the spleen" are probably cases of this disease. He emphasizes that pulmonary bilharziasis must be differentiated from the well known pulmonary symptoms that accompany bilharziasis of other organs.

Hospitalstidende, Copenhagen

78: 833-860 (Aug. 13) 1935

*Medical Treatment of Congenital Pyloric Stenosis. Elisabeth Svensgaard.—p. 833.
Secondary Pelagra: Review of Forty Cases. A. Hofman-Bang.—p. 845.
*Sporadic Goiter on Genotypical Basis. S. Hanum and J. E. Holst.—p. 855.

Treatment of Congenital Pyloric Stenosis. — Svensgaard's material consists of seventy-one patients, from 1911 to 1922, treated with stomach lavage and feeding by duodenal sound, forty-seven from 1922 to 1927 treated with atropine and sixty-one since 1927 treated with atropine methylnitrate. The average hospitalization was, respectively, 108, 96 and 77 days, the mortality 7.1, 6.4 and 1.6 per cent, and the average increase in weight per week 62, 91 and 122 Gm. The author emphasizes that treatment with salt solution is important until dehydration is past, when treatment with atropine or atropine methylnitrate is begun, and that the medicament is given one-half hour before meals. The grave prognosis for patients with a tendency to hemorrhage is stressed.

Sporadic Goiter on Genotypical Basis. — Hanum and Holst report ten cases of goiter in one family, in two generations, one in a man and nine in women or girls. They assert that these cases are definitely distinguished from exophthalmic goiter, although half of the patients have presented symptoms resembling those of exophthalmic goiter. While most of the reported cases of sporadic goiter on a genotypical basis have been nodose, only one of their instances is nodose, in one case information is lacking and in the remaining eight (a father and seven daughters) the goiter is diffuse. This difference, they suggest, may be partially explained by the different ages represented, the goiter noted in childhood, as in the seven sisters, most often being diffuse; but other factors are believed also to be present.